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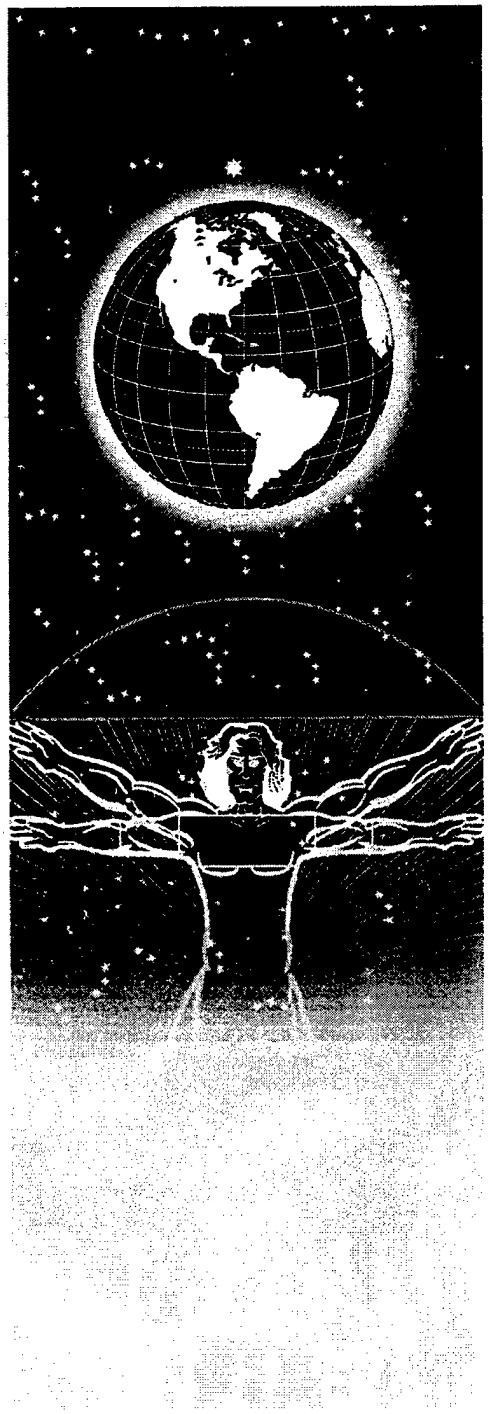

UNITED STATES AIR FORCE RESEARCH LABORATORY

RADIOFREQUENCY ELECTROMAGNETIC FIELDS (RFEMF) AND CANCER: A COMPREHENSIVE REVIEW OF THE LITERATURE PERTINENT TO AIR FORCE OPERATIONS

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This report has been reviewed and is approved for publication.

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13. ABSTRACT *(Maximum 200 words)*

Analyzed herein are the research studies on whether an association exists between the incidence or promotion of cancer and exposure to radiofrequency electromagnetic fields (RFEMF) in the nominal frequency region from 3 kHz to 300 GHz. Among the topics discussed are RFEMF and cancer in humans, including epidemiologic/occupational studies of populations actually exposed, or presumed to have been exposed to RFEMF, based on occupational titles or analogous considerations; in vivo and in vitro studies seeking cancer induction or promotion in mammals and mammalian tissues; and studies toward determining whether RFEMF is mutagenic or genotoxic to microorganisms or fruit flies. The findings of the studies on each topic are summarized in tabular form. Preceding those topics is a discussion of the need for using scientific criteria to assess the credibility of the findings of the various studies with regard to any potential risk to human health. Also presented are summaries of past and current exposure guidelines for human exposure to RFEMF. The overall conclusion of the analyses is that there is no scientifically valid basis for the existence of a causal linkage between RFEMF exposure and cancer incidence or promotion.

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RADIOFREQUENCY ELECTROMAGNETIC FIELDS (RFEMF) AND CANCER: A COMPREHENSIVE REVIEW OF THE LITERATURE PERTINENT TO AIR FORCE OPERATIONS

1 INTRODUCTION

1.1 GENERAL

This report is one of a planned series on various topics dealing with actual or potential biological effects of radiofrequency electromagnetic fields (RFEMF) and their possible consequences to human health. Specifically analyzed in this report are research papers (in English) on investigations that sought associations between the incidence of cancer and exposure to RFEMF. The critiques presented herein were derived predominantly from the peer-reviewed scientific literature on the topic.

The acronym "RFEMF" used herein refers to the emission and propagation of electromagnetic waves in the frequency range nominally from 3 kHz to 300 GHz (encompassing the radio frequency and microwave ranges). Such waves are characterized as nonionizing because the intrinsic (quantum) electromagnetic energy absorbed by a body at any frequency within those ranges is much too low to ionize (eject electrons) from molecules of the body. Other acronyms found in the bioeffects literature include "radiofrequency radiation" (RFR), the more general "electromagnetic fields" (EMF) and "electromagnetic radiation" (EMR). In recent years, however, the acronym EMF (or EMFs) has become associated frequently with potential biological effects of electric or magnetic fields (usually at 50 Hz or 60 Hz) from power lines and the conveyors of such power into machinery, appliances, and other devices within residences, businesses, and other establishments.

1.2 LITERATURE SELECTION

Some of the information herein was derived from reports previously prepared for, and issued by, the U.S. Air Force Research Laboratory (formerly Armstrong Laboratory), Brooks Air Force Base, Texas, such as Heynick (1987), Heynick and Polson (1996a), and Heynick and Polson (1996b). In addition, included are critiques of subsequently published papers and earlier papers not analyzed previously. With a few exceptions, presentations at scientific symposia or the abstracts of such presentations have not been analyzed, in the expectation that more detailed, peer-reviewed accounts of such studies will appear subsequently. Endeavors were made to analyze virtually all of the peer-reviewed papers published to date on this topic. It is likely, however, that some papers were missed. If so, the author of this report would be most appreciative of any suggested additions. Such papers, together with those published after this report is issued, may serve as the basis of a later update on this topic.

1.3 ASSESSMENT OF SCIENTIFIC INFORMATION AND RISK

A question often raised is why absolute guarantees cannot be given that any new RFEMF-emitting device, system, or other related technological advance will be totally harmless. From a strictly scientific viewpoint, it is basically impossible to prove with absolute certainty that any specific effect will or will not occur, a point especially applicable to biological sciences because of the enormous complexity and variety of life-based entities. Instead, the findings of high quality research, as critically judged by other researchers, are generally accepted with great confidence. The findings of additional research on the specific subject rarely tend to contradict those that have been accepted in critical review and published, but it can happen, hence the avoidance of absolute guarantees.

Experimental researchers generally describe their findings in terms of statistical probabilities based on the degree of validity of the methods used to study any specific effect and the validity of the techniques and accuracy of the instruments used to gather the data. An important point to recognize is that statistically based findings on the effects of a specific agent cannot be used to predict the outcome of exposing an individual to that agent. For example, the term "median effective dose" for an agent is defined as the dose that will produce the response to that agent in half of the subjects dosed. Before the dose is administered, however, how any specific individual will respond cannot be predicted, but the prediction that any subject will have a 50% chance of having the response is valid.

Ideally, the most direct evidence about possible deleterious effects of any RFEMF-emitting system on humans would necessitate performance of experiments in which human volunteers are exposed to the system's specific frequency range at the expected exposure levels and durations, with a variety of preselected biological endpoints to be evaluated. However, such data rarely if ever exist, and for ethical or pragmatic reasons, may not be obtainable. Instead, recourse is usually had to use of laboratory animals as surrogates for humans, a practice in wide use for assessing the possible effects of other agents. Thus, experimental evidence about RFEMF bioeffects is derived primarily from studies of laboratory animals that are exposed to RFEMF of specific characteristics, with appropriate quantification of their biological responses. However, it is important to note that the animals in such investigations generally have different anatomies and functional characteristics than humans, and that the RFEMF parameters and exposure durations may differ considerably from those of any specific RFEMF system of interest, usually necessitating extrapolation of the findings to humans.

Analysts disagree over whether the conventional scientific approach, in which an investigator finds a statistically significant difference in results (meaning that the probability of its occurrence by chance is very low) between experimental and control groups, can be applied toward considering whether the findings relate to humans. On the other hand, the researcher's statement that the differences between the groups are not statistically significant is not equivalent to the statement that there is no difference between the groups.

Regarding exposures of humans to RFEMF, some investigations on specific biological aspects have been done by exposing volunteers (with their informed consent), but for ethical reasons, very few of such studies have been conducted. Far more common are epidemiologic studies in which specific human populations have been exposed to environmental levels of RFEMF (and likely, to other physical or chemical agents as well). More generally, the aim of many epidemiologic studies is to assess the distribution of a disease or abnormal physiological condition in human populations and, to the extent feasible, to determine what factors or agents contribute importantly to the distribution (Lilienfeld and Lilienfeld, 1980).

Epidemiologic studies usually have far greater uncertainties than those with laboratory animals. The values of the exposure parameters (particularly exposure levels and durations) can vary widely with time for each individual and are highly variable from person to person, so accurate useful quantitative exposure data are rarely obtained. Also in many epidemiologic studies, the "exposed" group is selected by occupation, often with considerable uncertainty about the extent of exposure of not only the group of people presumed exposed to RFEMF but also of the control group selected for comparison and presumed not to have been exposed. In other studies, the groups to be compared are characterized as "high-exposure" and "low-exposure". Epidemiologists also try to select control groups that match the test groups in other factors besides RFEMF exposure, but the closeness and extent of such matching is often open to question.

Regarding studies with laboratory animals, several constraints can affect interpretation of the findings. Specifically, researchers disagree on whether an experimentally verified effect, especially one that is reversible or can be readily compensated for by the subject, constitutes a hazard. Also, the findings of some investigations, especially those that report new effects, rarely have been subjected to identical experiments by other investigators to confirm those findings. More often instead, another investigator may conduct analogous--but not identical--experiments to assess, clarify, or expand on the results of the previous study. The latter experiments ideally provide better means of incorporating the findings into the theory underlying the knowledge in a particular field of investigation, but may not necessarily confirm the results of the first investigation.

Conceivably, some agents may have biologically real effects so small in magnitude that the difference in mean response between the experimental and control groups may not be discernible within the scattering of values for both groups if the group sizes are small. Biological studies to detect such small differences and to indicate that they are statistically significant (to a prescribed probability that they are not due to chance) would require the use of large numbers of animals and, in some cases, long

exposure times. The expenditures in time and money to perform such studies may be so large that sponsoring institutions with limited budgets often decide that such studies are not cost-effective relative to the sponsor's overall objectives.

A frequent alternative approach is to predict effects at very low levels of an agent by extrapolation from findings at higher levels, on the basis of assumptions about the mathematical relationship between the level (or dose) of the agent and the degree of the effect. Such assumptions are often open to challenge, and this approach may lead to major disagreement over the possible existence of a threshold dose or dose rate below which the agent has no effects.

Scientific experiments are often directed toward the evaluation of only one factor. In the real world, however, interactions are far more complex. The synergistic effect of concurrent exposure to more than one factor is illustrated in a study of the incidence of lung cancer in uranium miners, in which the disease was found to be higher in the miners than in the general population, presumably because of the inhalation of radioactive material. However, the higher incidence in the nonsmoking miners was marginal, but those who smoked cigarettes had a much higher incidence of lung cancer than either nonsmoking miners or the general population.

It must be remembered that scientists also have personal values, goals, and attitudes. It often has been said that unbiased experts do not exist, because to become an accepted authority requires a long personal commitment to the chosen field that automatically leads to emphasis of certain viewpoints (not necessarily the same among those in that field). Thus, objectivity, like probabilistic scientific findings, may well be characteristic of scientists as a group but not necessarily applicable to any individual scientist. Personal biases among researchers in any study can consciously or subconsciously affect how an experiment is designed and how the data are interpreted. Particularly important is the way the findings are to be used in decision making, a process not necessarily involving the specific investigators, but others outside the field of expertise.

Assessment of the risk to human health from environmental agents (including RFEMF) and setting protection standards to reduce potential risk are extremely complex problems. In addition to technical and scientific questions, there are those of law, socioeconomics, and administration, often leading to risk-versus-benefit analyses. It is beyond the scope of this document to deal with those subjects in detail, but it is important that they be mentioned.

The subject of the existence or nonexistence of thresholds for noxious or deleterious effects of various agents has been debated at length. As a practical scientific matter, thresholds exist at least for some substances, because many naturally occurring substances are essential to life at specific concentrations and are toxic at higher concentrations (Horne, 1972). The existence of threshold RFEMF levels is considered in this report for various specific effects where such data are available.

1.4 RFEMF SAFETY GUIDELINES/STANDARDS

Terms such as "exposure standards" and "exposure guidelines" for human safety generally refer respectively to official directives (by federal, state, or municipal governmental agencies) and to non-government guidelines on maximum permissible exposure levels to electromagnetic fields. However, the two terms are often used interchangeably (even in the same document), so for simplicity, the word "guidelines" will be used herein for both. Maximum exposure levels for the general public are often lower than for persons in occupations and/or working areas where they may be exposed periodically to higher than background levels of such electromagnetic fields. In both situations, the levels are usually expressed as maximum permissible incident field intensities and/or power densities in specific frequency ranges for stated exposure durations.

Guidelines for exposure to electromagnetic fields have been developed by several U.S.-based and international organizations for occupational use and/or the general public. Such organizations include the American National Standards Institute (ANSI), the Institute of Electrical and Electronics Engineers (IEEE), the National Council on Radiological Protection and Measurements (NCRP), the American Conference of

Governmental Industrial Hygienists (ACGIH), and the International Commission on Non-Ionizing Radiation Protection (ICNIRP). In some instances, agencies of the federal government such as the Federal Communications Commission (FCC), the Federal Occupational Safety and Health Administration (OSHA), and Department of Defense agencies had adopted such guidelines as enforceable standards, and various state and municipal bodies had adopted more stringent versions thereof.

In most guidelines for human exposure to RFEMF, the maximum permissible exposures (MPEs) are stated in terms of the maximum allowable incident power densities, expressed in milliwatts per square centimeter (mW/cm^2) or in watts per square meter (W/m^2, with 1 W/m^2 = 0.1 mW/cm^2). Such MPEs are selected on the basis of the highest values of "specific absorption rate" (SAR) that were found to be not harmful to animals in experimental studies. The "local SAR" within a body is defined as the rate at which RFEMF energy is absorbed in any small volume of that body (usually 1 cubic centimeter). It is usually expressed in watts per kilogram (W/kg) of the mass in that volume (or sometimes in milliwatts per gram, with 1 mW/g = 1 W/kg). For any specific value of incident power density, the local SAR thus defined varies with location within the body. (See a more detailed discussion of SAR in Section 2.3.)

The internal spatial variations of local SAR are difficult to determine for most complex bodies, so the term "whole-body SAR" is often used to represent the spatially averaged value of SAR for the whole body. The whole-body SAR is a quantity that can be measured (for example, by calorimetry) without requiring knowledge of the spatial variations of local SAR. The term "partial-body" SAR is used in appropriate cases, such as when absorption of RFEMF occurs primarily in a specific region of the body due to exposure from a nearby emitter (a hand-held transmitter for example).

The values of local and whole-body SARs depend on the frequencies of the incident field and other properties of the body, such as its size, shape, internal constituents, and orientation relative to the incoming fields. For certain specified exposure conditions, such as at frequencies below about 300 MHz and/or within the "near-field" distance of an antenna that is emitting electromagnetic energy, it is usually necessary to consider the electric and magnetic fields separately. For such conditions, exposure guidelines are expressed in terms of maximum allowable electric fields in volts per meter (V/m) and maximum allowable magnetic fields in amperes per meter (A/m). Such guidelines are also based on whole-body or partial-body SARs.

Some organizations that developed guidelines for occupational exposure and for exposure of the general public usually reexamine such guidelines for possible reaffirmation or revisions on a cyclic schedule. As an example, the American National Standards Institute had published a set of guidelines in 1974 (ANSI, 1974) applicable both to occupational exposure and exposure of the general public. Such exposures were not to exceed 10 mW/cm^2 or approximate equivalent electric and magnetic field strengths (200 V/m and 0.5 A/m) independent of frequency over the range from 10 MHz to 100 GHz. The federal Occupational Safety and Health Administration (OSHA), which had promulgated the 10-mW/cm^2 level as a voluntary occupational standard, later found that the standard was not legally enforceable.

In 1982, ANSI issued a revision (ANSI, 1982) of those 1974 guidelines, also applicable to both occupational and general-public exposure, based on critical analyses of the then current scientific knowledge about biological effects of RFEMF. The highest permissible incident field strengths or equivalent power densities were based on a maximum whole-body SAR of 4 W/kg, at or above which detrimental effects had been observed in experimental studies with laboratory animals. A safety factor of 10 was provided in the 1982 ANSI guidelines, yielding a maximum allowable SAR of 0.4 W/kg. At a fixed SAR (such as 0.4 W/kg), the maximum allowable incident electric field, magnetic field, and equivalent power densities are frequency-dependent; the frequency range covered by ANSI (1982) was 300 kHz to 100 GHz. As in ANSI (1974), the limits in ANSI (1982) were not to be exceeded for exposures averaged over any 0.1-hour period. At SAR=0.4 W/kg, the smallest maximum allowable incident power density was 1 mW/cm^2 for the subrange 30-300 MHz in which RFEMF absorption by the human body as a resonant entity (much like a dipole antenna) is highest.

In 1988, the functions of the various ANSI subcommittees on developing guidelines for RFEMF exposure were transferred to Subcommittee IV of Standards Coordinating Committee (SCC) 28, a then

new body under the jurisdiction of the Institute of Electrical and Electronics Engineers (IEEE). That body developed a revision of the 1982 ANSI guidelines based on selecting and analyzing the important research papers in a then updated database of the literature on biological effects of RFEMF. The revision, titled "IEEE Standard for Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 3 kHz to 300 GHz," was approved by the IEEE in 1991 and was published in 1992. It was also approved by ANSI in 1992, and is cited herein as the ANSI/IEEE (1992) guidelines.

As in ANSI (1982), the ANSI/IEEE (1992) guidelines, expressed in terms of maximum permissible exposures (MPEs), are largely based on 0.4 W/kg (4 W/kg reduced by a safety factor of 10). Those guidelines cover the frequency range from 3 kHz to 300 GHz (instead of 300 kHz to 100 GHz). They separately specify MPEs in "uncontrolled environments" (areas accessible by the general population) and "controlled environments" (areas where those entering know or are made aware of the presence of such fields, such as in occupational situations). The guidelines for controlled environments and uncontrolled environments are displayed in Tables 1 and 2, respectively.

TABLE 1: MAXIMUM PERMISSIBLE EXPOSURE LIMITS FOR CONTROLLED ENVIRONMENTS*
[ANSI/IEEE (1992)]

Frequency Range (MHz)	E (V/m)	H (A/m)	Power Density, S (mW/cm^2)	Averaging Time E^2, H^2, or S (minutes)
0.003 - 0.1	614	163	(100; 1,000,000)	6
0.1 - 3.0	614	16.3 /f	(100; $10,000/f^2$)	6
3 - 30	1,842 /f	16.3 /f	($900/f^2$; $10,000/f^2$)	6
30 - 100	61.4	16.3 /f	1.0; $10,000/f^2$)	6
100 - 300	61.4	0.163	1.0	6
300 – 3,000			f /300	6
3,000 – 15,000			10	6
15,000 – 300,000			10	$(616,000)/f^{1.2}$

TABLE 2: MAXIMUM PERMISSIBLE EXPOSURE LIMITS FOR UNCONTROLLED ENVIRONMENTS*
[ANSI/IEEE (1992)]

Frequency Range MHz)	E (V/m)	H (A/m)	Power Density, S (mW/cm^2)	Averaging Time E^2, S or H^2 (minutes)	
0.003 - 0.1	614	163	(100; 1,000,000)	6	6
0.1 - 1.34	614	16.3 /f	(100; $10,000/f^2$)	6	6
1.34 - 3.0	823.8/f	16.3 /f	($180/f^2$; $10,000/f^2$)	$f^2/0.3$	6
3 - 30	823.8/f	16.3 /f	($180/f^2$; $10,000/f^2$)	30	6
30 - 100	27.5	$158.3/f^{1.668}$	($0.2; 940,000/f^{3.336}$)	30	$0.0636f^{1.337}$
100 - 300	27.5	0.0729	0.2	30	30
300 – 3,000			f /1500	30	
3,000 – 15,000			f /1500	90,000/f	
15,000 – 300,000			10	$(616,000)/f^{1.2}$	

*In both tables above, the exposure values in terms of electric and magnetic field strengths are the values obtained by spatially averaging values over a plane area equivalent to a vertical cross section of the human body (the projected area) facing the RFEMF source.

**The equivalent plane-wave power densities for E (V/m) and H (A/m), expressed in W/m^2, can be calculated from the equations $S = E^2/377$ and $S = 377H^2$, where 377 (ohms) is the impedance of free space. To obtain the corresponding values of S in mW/cm^2 as shown in the tables, divide the values expressed in W/m^2 by 10. Note that even though such calculated equivalent power densities may not be

appropriate for near-field conditions, they are useful for comparing them with the power density limits for the higher frequency ranges.

For controlled environments, those guidelines also specify the following limits on pulsed RFEMF:

In the frequency range 0.1 to 300,000 MHz, the peak temporal MPE, expressed in terms of the electric field, is 100 kV/m. For single RFEMF pulses of duration less than 100 milliseconds in this frequency range, the peak MPE is given by the E-field equivalent power-density MPE of Table 1 in the formula:

Peak MPE = MPE x averaging time / (5 x pulse duration).

A maximum of five such pulses, with a pulse repetition period of at least 100 milliseconds, is permitted during any period equal to the averaging time. For pulse trains of more than 5 pulses or for pulse durations that exceed 100 milliseconds, the following formula applies for averaging over any 100-millisecond period:

Sum of Peak MPE x pulse duration = MPE x averaging time / 5.

Also contained in the ANSI/IEEE (1992) guidelines are the maximum allowable values of radiofrequency current induced within the feet of a person immersed in a radiofrequency field, or by physical contact of a person with an inanimate object (such as a fence or vehicle) that has become electrically charged by immersion in a radiofrequency field. The limits for controlled and uncontrolled environments, shown respectively in Tables 3 and 4, are applicable only within the frequency range from 3 kHz to 100 MHz (where such effects can occur).

TABLE 3: LIMITS ON INDUCED AND CONTACT CURRENTS IN CONTROLLED ENVIRONMENTS
[ANSI/IEEE (1992)]

Frequency Range (MHz)	Maximum Current Through Both Feet (mA)	Maximum Current Through Each Foot (mA)	Contact
0.003-0.1	2,000f	1,000f	1,000f
0.1-100	200	100	100

TABLE 4: LIMITS ON INDUCED AND CONTACT CURRENTS IN UNCONTROLLED ENVIRONMENTS
[ANSI/IEEE (1992)]

Frequency Range (MHz)	Maximum Current Through Both Feet (mA)	Maximum Current Through Each Foot (mA)	Contact
0.003-0.1	900f	450f	450f
0.1-100	90	45	45

The ANSI/IEEE (1992) guidelines also specify exclusions for exposures, under controlled and uncontrolled environments, from devices that emit RFEMF of low power, e.g., 7 watts or less in the frequency range 100 kHz to 450 MHz in a controlled (user-aware) environment.

In 1997, SCC-28 formally reaffirmed the ANSI/IEEE (1992) guidelines. Those guidelines are currently undergoing the cyclic review process.

In 1986, Scientific Committee 53 of the National Council on Radiation Protection and Measurements (NCRP) issued its guidelines (NCRP, 1986), which covered the frequency range from 300 kHz to 100 GHz. Those exposure limits were also based on 4 W/kg, but with a safety factor of 50 recommended for the general population instead of 10. The corresponding lowest power-density limit was 0.2 mW/cm^2. The limits specified in ANSI (1982) [with the safety factor of 10] were recommended for occupational exposure. The fivefold larger safety factor for the general population was based on the assumption that

the general public is exposed continuously (168 hours per week) and that the ratio of 40 hours in the work week to 168 hours is approximately 0.2. Those guidelines are also undergoing cyclic reexamination.

Well over a decade ago, the Environmental Protection Agency (EPA) had been planning to issue RFEMF exposure guidelines for the general population. The rationale for those guidelines was to be based on a literature review by Elder and Cahill (1984). On 30 July 1986, the EPA had published a Notice of Proposed Recommendations (EPA, 1986a) in which three options based on risk, benefit, and cost analyses were proposed for comment: a tenfold, fivefold, or no reduction from 0.4 W/kg (i.e., to 0.04, 0.08, or 0.4 W/kg). However, the EPA subsequently had decided not to issue such exposure guidelines for the general population, and it has not done so to date.

On 26-27 April 1993, the EPA conducted a workshop in Bethesda, MD, to review the current status of research on biological effects of RFEMF. Invited experts gave formal presentations on various topics, including RFEMF dosimetry, magnetic resonance imaging (MRI), thermal physiology, epidemiology, and a general review of the RFEMF-bioeffects literature. The formal presentations were followed by workshops on those and other topics, and then by summaries at a plenary session. Last, the Chair of the plenary session (a member of EPA's Science Advisory Board) proposed the following two-part motion, which was overwhelmingly approved by those present:

1) That the EPA resume its activities on RFEMF-bioeffects research with the intent of establishing a national standard for RFEMF exposure.

2) That the EPA in the interim, adopt the ANSI/IEEE (1992) standard.

In 1995, the EPA stated its intention to issue guidelines for RFEMF exposure of the general public, guidelines that the EPA said would be based solely on thermal effects. The EPA commented that not enough is known about long-term effects of exposure to "non-thermal" RFEMF levels to use them as a basis for setting standards. In 1996, however, EPA abandoned this effort.

In the absence of a governing Federal standard (but not necessarily for that reason), various state, county, and municipal bodies promulgated ordinances on exposure of the general population to RFEMF, some of which were more stringent than those of ANSI (1982) or ANSI/IEEE (1992). Most such standards refer to the 4-W/kg SAR used as the basis of those 1982 and 1992 guidelines, but with a reduction factor for safety of 50 [to 0.08 W/kg] or larger instead of 10.

In 1985, the Federal Communications Commission (FCC) adopted the ANSI (1982) guidelines as a standard that FCC licensees had to meet, thus rendering it a mandatory rather than a voluntary federal standard. On 8 April 1993, the FCC issued a "NOTICE OF PROPOSED RULE MAKING" (FCC, 1993) to consider adoption of the ANSI/IEEE (1992) standard to replace the ANSI (1982) standard. In that notice, the FCC requested comments and recommendations from various federal, state, and local municipal bodies; industrial organizations, including wireless communications businesses; and committees engaged in preparing or revising exposure guidelines.

In a "REPORT AND ORDER" dated 1 August 1996 (FCC, 1996a) the FCC noted the receipt of more than 100 responses. Those responses included a recommendation from the EPA that the FCC adopt a mixture of the exposure limits in the NCRP (1986) document and those in the ANSI/IEEE (1992) standard, to the extent such limits pertain to its jurisdictional areas. The FCC stated its acceptance of most but not all of the EPA recommendations. The stated basis for including the exposure limits in NCRP (1986) was that where they differ from those in ANSI/IEEE (1992), they would be more protective of the general public. Because of the lack of supporting evidence for this point, its validity was questioned in opposing responses submitted by various other organizations and individuals (including agencies in the Department of Defense), responses cited but not discussed in FCC (1996a).

Thus, as detailed in FCC (1996a), that agency adopted the guidelines for occupational/controlled and general-population/uncontrolled exposures shown respectively in Tables 5A and 5B.

TABLE 5A: MAXIMUM PERMISSIBLE LIMITS FOR OCCUPATIONAL/CONTROLLED EXPOSURE TO RFEMF
[FCC (1996a)]

Frequency f (MHz)	E (V/m)	H (A/m)	PD_{eq}* (mW/cm^2)	Averaging Time (minutes)
0.3-3.0	614	1.63	100	6
3.0-30	1,842/f	4.89/f	$900/f^2$	6
30-300	61.4	0.163	1.0	6
300-1,500	--	--	f/300	6
1,500-100,000	--	--	5	6

*PD_{eq} = Free-space equivalent power density.

TABLE 5B: MAXIMUM PERMISSIBLE LIMITS FOR GENERAL-POPULATION/UNCONTROLLED EXPOSURE TO RFEMF
[FCC (1996b)]

Frequency f (MHz)	E (V/m)	H (A/m)	PD_{eq}* (mW/cm^2)	Averaging Time (minutes)
0.3-1.34	614	1.63	100	30
1.34-30	824/f	2.19/f	$180/f^2$	30
30-300	27.5	0.073	0.2	30
300-1,500	--	--	f/1500	30
1,500-100,000	--	--	1.0	30

*PD_{eq} = Free-space equivalent power density.

Among the major similarities and differences between the FCC (1996a) and ANSI/IEEE (1992) standards are:

1) Both standards are largely based on a whole-body SAR of 4 W/kg, reduced by specific safety factors.

2) The FCC (1996a) standard only covers the frequency range from 300 kHz to 100 GHz, whereas the frequency range of the ANSI/IEEE (1992) standard is from 3 kHz to 300 GHz.

3) In both standards, the lowest maximum permissible incident free-space power density for occupational/controlled exposures is 1 mW/cm^2 (reduction factor of 10) for the frequency range 100-300 MHz, with an averaging time of 6 minutes.

4) Similarly in both standards, the lowest maximum permissible incident free-space power density for general-public/uncontrolled exposures is 0.2 mW/cm^2 (reduction factor of 50) for the same frequency range, with an averaging time of 30 minutes.

5) In both standards, the MPEs for occupational/controlled exposures in the frequency range 300-1500 MHz are the same: f/300. The MPEs for the frequency range 1500-3000 MHz in the ANSI/IEEE (1992) standard are also given by f/300, rising to 10 mW/cm^2 at 3.0 GHz, and remaining at that value to 300 GHz. However, the MPEs in FCC (1996a) rise to only 5 mW/cm^2 at 1.5 GHz and remain at that value to 100 GHz [the upper frequency limit of FCC (1996a)].

6) For general-public/uncontrolled exposures in the frequency range 3000-15000 MHz [3-15 GHz], the ANSI/IEEE (1992) standard prescribes the formula f/1500, yielding 10 mW/cm^2 at 15 GHz, a value also specified for the range 15-300 GHz. In the FCC (1996a) standard, the formula f/1500 is applicable only in the range 300-1500 MHz, rising to 1.0 mW/cm^2 at 1.5 GHz and remaining at that value to 100 GHz.

7) By contrast with the point in FCC (1996a) about the NCRP (1986) providing more protection to the general public, the averaging times in ANSI/IEEE (1992) for general-public/uncontrolled exposures in the frequency range 3.0-100 GHz are progressively shorter than 30 minutes with increasing frequency. Thus, the MPEs of ANSI/IEEE (1992) are more stringent than those recommended in FCC (1996a). At 15 GHz, for example, the total maximum permissible energy density equivalent to 5 mW/cm^2 for 30 minutes (FCC, 1996a) is 9 J/cm^2, whereas for 10 mW/cm^2 for 6 minutes (ANSI/IEEE, 1992), it is only 3.6 J/cm^2. Such total-energy calculations are pertinent primarily to incident RFEMF in the form of very short individual pulses of extremely high peak power density. In such cases, the corresponding average power densities per pulse would be inversely proportional to the averaging time.

Formal protests about adoption of the exposure standard contained in the FCC (1996a) document were filed by various organizations, including agencies of the Department of Defense (DoD). Those protests presumably were primarily toward the lack of scientific justification for the FCC preference in the use of the 10-year-old NCRP (1986) document over the ANSI/IEEE (1992) standard [where they differ] and were against alleged improper procedures used in the adoption of the new FCC standard.

On 24 December 1996, the FCC issued a "FIRST MEMORANDUM OPINION AND ORDER" (FCC, 1996b), in which the transition period was extended for applicants and station licensees to determine compliance with the new requirements for evaluating the environmental effects of radiofrequency (RF) electromagnetic fields from FCC-regulated transmitters. For most radio services, the transition period was extended by eight months, to September 1, 1997. For the Amateur Radio Service, it was extended until January 1, 1998. The memorandum also allowed changes to amateur radio operator license examinations to be made as the examinations were routinely revised up to July 1, 1998.

The FCC adopted the "hybrid" standard above to include RFEMF exposures from the rapidly burgeoning cellular telephone industry, satellite communications, and the move toward advanced digital technology. In doing so, it stated unequivocally that it has jurisdiction in promulgating federal RFEMF exposure standards for all transmitters not operated by the federal government. (The FCC does license all transmitters used locally, such as by fire and police departments, ambulances, water districts, and so forth.)

The National Telecommunications and Information Administration (NTIA) of the Commerce Department coordinates the assignment and use of the various portions of the frequency spectrum for all federal governmental agencies, including the DoD. The RFEMF exposure standards for transmitters directly operated by agencies of the DoD are promulgated by those agencies, based on Department of Defense Instruction DoDI 6055.11 (DoDI, 1995). Among those standards is the Air Force Occupational Safety and Health Standard 48-9 dated 1 August 1997, "Radio Frequency Radiation (RFR) Safety Program" (AFOSH, 1997), which contains the permissible exposure limits (PELs) "used throughout industry [?]", as instructed in DoDI 6055.11. As in the ANSI/IEEE (1992) guidelines, the PELs are specified separately for controlled and uncontrolled environments based on a SAR of 4 W/kg, with respective reduction factors of 10 (to 0.4 W/kg) and 50 (to 0.08 W/kg). Those PELs are shown in Tables 6A and 6B.

TABLE 6A: PELS FOR CONTROLLED ENVIRONMENTS
[AFOSH (1997)]

A: RFR Fields

Frequency Range f (MHz)	Electric Field E (V/m)	Magnetic Field H (A/m)	Power Density S (mW/cm^2) (E,H)	Averaging Time T_{avg} (min) (E,H,S)
0.003 - 0.1	614	163	$(10^2, 10^6)$	6
0.1 - 3.0	614	16.3/f	$(10^2, 10^4/f^2)$	6
3 - 30	1842/f	16.3/f	$(900/f^2, 10^4/f^2)$	6
30 - 100	61.4	16.3/f	$(1.0, 10^4/f^2)$	6
100 - 300	61.4	0.163	1.0	6
300 – 3,000			F/300	6
3,000 – 15,000			10	6
15,000 – 300,000			10	$616{,}000/f^{1.2}$

B: RFR Induced and Contact Current Restrictions

Frequency Range (MHz)	Maximum Current Through Both Feet (mA)	Maximum Current Through Each Foot (mA)	Contact Current (mA)
0.003 - 0.1	2,000f	1,000f	1,000f
0.1 - 100	200	100	100

C: Pulsed RFR Fields (apply only when there are less than 5 pulses within the averaging time)

Frequency Range f (MHz)	Peak Electric Field E (kV/m)	Pulse For Peak Power Density Pulse Durations < 100 msec (mW/cm^2)
0.1 – 300,000	100	$(PEL)(T_{avg})/(5)$(pulse width)

D: Partial-Body Exposures

Frequency Range f (MHz)	Peak Value of Mean Squared Field (V^2/m^2 or A^2/m^2)	Equivalent Power Density (mW/cm^2)
0.1 - 300	$< 20\ E^2$ or $20\ H^2$	
300 – 6,000		< 20
6,000 – 96,000		$< 20(f/6{,}000)^{0.25}$
96,000 – 300,000		40

TABLE 6B: PELS FOR UNCONTROLLED ENVIRONMENTS
[AFOSH (1997)]

A: RFR Fields

Frequency Range f (MHz)	Electric Field E (V/m)	Magnetic Field H (A/m)	Power Density S (mW/cm^2) (E,H)	T_{avg} (min) (E,S)	(H)
0.003 - 0.1	614	163	$(10^2, 10^6)$	6	6
0.1 - 1.34	614	16.3/f	$(10^2, 10^4/f^2)$	6	6
1.34 - 3.0	823.8/f	16.3/f	$(180/f^2, 10^4/f^2)$	$f^2/0.3$	6
3 - 30	823.8/f	16.3/f	$(180/f^2, 10^4/f^2)$	30	6
30 - 100	27.5	$158.3/f^{1.668}$	$(0.2, 9.4\times10^5/f^{3.336}$	30	$0.0636f^{1.337}$
100 - 300	27.5	0.0729	0.2	30	30
300 – 3,000			f/1,500	30	
3,000 – 15,000			f/1,500	90,000/f	
15,000 – 300,000			10	$616{,}000/f^{1.2}$	

B: RFR Induced and Contact Current Restrictions

Frequency Range (MHz)	Maximum Current Through Both Feet (mA)	Maximum Current Through Each Foot (mA)	Contact Current (mA)
0.003 - 0.1	900f	450f	450f
0.1 - 100	90	45	45

C: Pulsed RFR Fields (apply only when there are less than 5 pulses within the averaging time)

Frequency Range f (MHz)	Peak Electric Field E (kV/m)	Pulse For Peak Power Density Pulse Durations < 100 msec (mW/cm^2)
0.1 – 300,000	100	$(PEL)(T_{avg})/(5)$(pulse width)

D: Partial-Body Exposures

Frequency Range f (MHz)	Peak Value of Mean Squared Field (V^2/m^2 or A^2/m^2)	Equivalent Power Density (mW/cm^2)
0. 1- 300	< 20 E^2 or 20 H^2	
300 – 6,000		4
6,000 – 96,000		f/1,500
96,000 – 300,000		20

Note: Measurements to determine adherence to the PEL shall be made at distances of 20 cm or greater from any object.

AFOSH (1997) also includes PELs for high power microwave (HPM) systems and electromagnetic pulse (EMP) systems.

The worldwide web (www) site http://afpubs.hq.af.mil is referenced in the document for those who wish to obtain copies thereof.

Other organizations in the U.S.A., in other countries, and multinational bodies, have issued exposure guidelines. One such multinational body was the International Non-Ionizing Radiation Committee (INIRC) of the International Radiation Protection Association (IRPA), with participants from Australia, France, Federal Republic of Germany, Italy, the Netherlands, Sweden, the U.K., and the U.S. IRPA/INIRC (1988) had published guidelines for occupational and general-public exposure to RFEMF for the frequencies in the range 0.1 MHz to 300 GHz. The occupational exposure limits in the range 10 MHz upward were based on a whole-body SAR of 0.4 W/kg, and were fivefold lower (based on 0.08 W/kg) for the general public. The environmental health criteria issued by the World Health Organization (WHO, 1981) served as the rationale.

The IRPA/INIRC (1988) guidelines also specified a maximum body-to-ground current of 200 mA and recommended that for pulsed RFEMF, the instantaneous peak values should not exceed 100 times the 0.1-hour-averaged limits at all frequencies. Regarding shocks and burns, the guidelines stated: "Hazards of RF burns should be eliminated by limiting currents from contact with metal objects. In most situations this may be achieved by reducing the E values from 614 to 194 V/m in the range from 0.1 to 1 MHz and from 614/f to 194/f in the range from >1 to 10 MHz. In general, RF burns will not occur from currents on point contact of 50 mA or less." Regarding pulsed RFEMF, the guidelines suggested that the pulse power density (averaged over the pulse duration) should not exceed 1000 times the specified average planewave power density limits, or that the peak field strengths not exceed 32 times the specified field strengths.

In 1990, IRPA/INIRC completed a major revision in conjunction with a WHO Task Group. The criteria were published (WHO, 1993), but the revised guidelines were not issued that year. In May 1992, the International Commission on Non-Ionizing Radiation Protection (ICNIRP) was established as the successor to IRPA/INIRC. Among the latter's activities was issuance of a statement (ICNIRP, 1996) regarding recommended limits for exposure to the RFEMF from hand-held radiotelephones and base transmitters in the nominal frequency range from 800 MHz to 2 GHz. The statement included

recommendations that the local SAR in the head not exceed 10 W/kg for occupational exposure or 2 W/kg for general-public exposure, both averaged over any 10 grams of tissue.

The new organization recently issued a set of exposure guidelines that cover the frequency spectrum from 0 to 300 GHz [ICNIRP (1998)]. The development of the guidelines was based on reviews of a number of topics, including various epidemiologic studies, investigations with human volunteers, experiments with laboratory animals, and studies of various tissue and cellular preparations. Tables 7A, 7 B, 8A, and 8B below (adapted from Tables 4, 6, 7, and 8 of the paper) display the major exposure limits on electric and magnetic fields, and near-field and far-field electromagnetic radiations specified in the guidelines for specific regions of the frequency spectrum above.

TABLE 7A: BASIC RESTRICTIONS FOR TIME VARYING ELECTRIC AND MAGNETIC FIELDS FOR FREQUENCIES UP TO 10 GHz
[ICNIRP (1998)]

Frequency Range	Current Density for Head &Trunk (mA/m^2, rms)	Whole-Body Average SAR (W/kg)	Localized SAR Head & Trunk (W/kg)	Localized SAR Limbs (W/kg)
		OCCUPAT. EXPOSURE		
Up to 1 Hz	40	--	--	--
1-4 Hz	40/f	--	--	--
4 Hz-1 kHz	10	--	--	--
1-100 kHz	f/100	--	--	--
100 kHz-10 MHz	f/100	0.4	10	20
10 MHz-10 GHz	--	0.4	10	20
		GEN.-PUBLIC EXPOSURE		
Up to 1 Hz	8	--	--	--
1-4 Hz	8/f	--	--	--
4 Hz-1 kHz	2	--	--	--
1-100 kHz	f/500	--	--	--
100 kHz-10 MHz	f/500	0.08	2	4
10 MHz-10 GHz	--	0.08	2	4

Notes:
1. f is the frequency in Hz.
2. Because of electrical inhomogeneity of the body, current densities should be averaged over a cross-section of 1 cm^2 perpendicular to the current direction.
3. For frequencies up to 100 kHz, peak current density values can be obtained by multiplying the rms value by $\sqrt{2}$ (~1.414). For pulses of duration t_p, the equivalent frequency to apply in the basic restrictions should be calculated as $f = 1 / (2t_p)$.
4. For frequencies up to 100 kHz and for pulsed magnetic fields, the maximum current density associated with the pulses can be calculated from the rise/fall times and the maximum rate of change of magnetic flux density. The induced current density can then be compared with the appropriate basic restriction.
5. All SAR values are to be averaged over any 6-minute period.
6. Localized SAR averaging mass is any 10 grams of contiguous tissue; the maximum SAR so obtained should be the value used for the estimation of exposure.
7. See 2nd sentence of 3. Additionally, for pulsed exposures in the frequency range 0.3 to 10 GHz and for localized exposure of the head, in order to limit or avoid auditory effects caused by thermoelastic expansion, an additional basic restriction is recommended. This is that the SA should not exceed 10 mJ/kg for workers and 2 mJ/kg for the general public, averaged over 10 grams of tissue.

For frequencies in the range 10-300 GHz, the basic power-density restrictions are 50 W/m^2 for occupational exposure and 10 W/m^2 for exposure of the general public. Those levels that should be averaged over any 20 cm^2 of exposed area and any $68/f^{1.05}$ -minute period (where f is in GHz) to compensate for progressively shorter penetration depth with increasing frequency. Also, spatial maximum power densities, averaged over 1 cm^2 should not exceed 20 times these values.

TABLE 7B: REFERENCE LEVELS FOR OCCUPATIONAL EXPOSURE TO TIME-VARYING ELECTRIC AND MAGNETIC FIELDS (UNPERTURBED RMS VALUES)
[ICNIRP (1998)]

Frequency Range	E-Field Strength (V/m)	H-Field Strength (A/m)	B-Field (μT)	Equiv. Plane-Wave Power Density S_{eq} (W/m^2)
Up to 1 Hz	---	1.63×10^5	2×10^5	---
1-8 Hz	20,000	$1.63 \times 10^5 / f^2$	$2 \times 10^5 / f^2$	---
8-25 Hz	20,000	$2 \times 10^4 / f$	$2.5 \times 10^4 / f$	---
0.025-0.82 kHz	500/f	20/f	25/f	---
0.82-65 kHz	610	24.4	30.7	---
0.065-1MHz	610	1.6/f	2.0/f	---
1-10 MHz	610/f	1.6/f	2.0/f	---
10-400 MHz	61	0.16	0.2	10
400-2,000 MHz	$3f^{0.5}$	$0.008f^{0.5}$	$0.01f^{0.5}$	f/40
2-300 GHz	137	0.36	0.45	50

Notes:
1. f as indicated in the frequency range column.
2. Provided that basic restrictions are met and adverse indirect effects can be excluded, field strength values can be exceeded.
3. For frequencies between 100 kHz and 10 GHz, E^2, H^2, B^2, and S_{eq} are to be averaged over any 6-minute period.
4. For peak values at frequencies up to 100 kHz, see note 3 of the previous table.
5. For peak values at frequencies exceeding 100 kHz, see Figs. 1 and 2 of the paper. Between 100 kHz and 10 MHz, peak field strengths are obtained by interpolation from the 1.5-fold peak at 100 kHz to the 32-fold peak at 10 MHz. For frequencies exceeding 10 MHz, it is suggested that the peak equivalent plane wave power density, as averaged over the pulse width, does not exceed 1,000 times the S_{eq} restrictions, or that the field strength does not exceed 32 times the field strength exposure levels given in the table.
6. For frequencies exceeding 10 GHz, E^2, H^2, B^2, and S_{eq}, are to be averaged over any $68/f^{1.05}$-minute period (f in GHz).
7. No electric-field values are provided for frequencies less than 1Hz, which are effectively static electric fields. Electric shock from low-impedance sources are prevented by establishing electric safety procedures for such equipment.

TABLE 8A: REFERENCE LEVELS FOR GENERAL PUBLIC EXPOSURE TO TIME-VARYING ELECTRIC AND MAGNETIC FIELDS (UNPERTURBED RMS VALUES)
[ICNIRP (1998)]

Frequency Range	E-Field Strength (V/m)	H-Field Strength (A/m)	B-Field (μT)	Equiv. Plane-Wave Power Density $S_{eq.}$ (W/m^2)
Up to 1 Hz	---	3.2×10^4	4×10^4	---
1-8 Hz	10,000	$3.2 \times 10^4 / f^2$	$4 \times 10^4 / f^2$	---
8-25 Hz	10,000	4,000/f	5,000/f	---
0.025-0.8 kHz	250/f	4/f	5/f	---
0.8-3 kHz	250/f	5	6.25	---
3-150 kHz	87	5	6.25	---
0.15-1MHz	87	0.73/f	0.092/f	---
1-10 MHz	$87/f^{0.5}$	0.73/f	0.092/f	---
10-400 MHz	28	0.073	0.092	2
400-2,000 MHz	$1.375f^{0.5}$	$0.0037f^{0.5}$	$0.0046f^{0.5}$	f/200
400-2,000 MHz	61	0.16	0.20	10

Notes:
1-6: The same as those in the previous table.
7: No E-field value is provided for frequencies less than 1 Hz, at which E-fields are effectively static electric fields. Surface electric charges will not be perceived at field strengths less than 25 kV/m. Spark-discharges that cause stress or annoyance should be avoided.

TABLE 8B: REFERENCE LEVELS FOR TIME VARYING CONTACT CURRENTS FROM CONDUCTIVE OBJECTS
[ICNIRP (1998)]

Exposure	Frequency Range	Maximum Contact Current (mA)
Occupational:	Up to 2.5 kHz	1.0
	2.5-100 kHz	0.4f
	100 kHz-110 MHz	40
General:	Up to 2.5 kHz	0.5
	2.5-100 kHz	0.2f
	100 kHz-110 MHz	20

A point of some relevance to this report on cancer and RFEMF exposure is that the ICNIRP (1998) document also cited studies that sought or reported a possible link between cancer incidence and exposure to the much lower powerline frequencies than those of RFEMF. It also cited the recent report by the National Academy of Science/National Research Council (NAS, 1996) about no clear evidence for such a link. The view expressed in ICNIRP (1998) [p. 499] is that "the results from the epidemiologic research on EMF field exposure and cancer, including childhood leukemia, are not strong enough in the absence of support from experimental research to form a scientific basis for setting exposure guidelines."

Perhaps still of interest are the exposure standards of the U.S.S.R. (Czerski, 1985) before its transformation into a confederation of independent republics. For frequencies below 300 MHz, the exposure limits (ELs) were given separately for the E-field and H-field. For the range 300 MHz to 300 GHz, the concept "permissible energy load" or allowable product of incident power density and exposure duration (PT) was used in the occupational standard, subject to a maximum power density of 1 mW/cm^2. The 1984 occupational ELs and PTs are displayed in Table 9.

TABLE 9: U.S.S.R. OCCUPATIONAL STANDARD FOR EXPOSURE TO RFEMF (1984)
[Czerski, (1985)]

Frequency (MHz)	E-Field EL (V/m)	H-Field EL (A/m)
0.06-1.5	50	5
1.5-3	50	*
3-3	20	*
30-50	10	0.3
50-30	5	*
300-300,000	**	*

*No EL specification for H-field in this frequency range.
**PT = 2 W.hr/m^2 for stationary fields; PT = 20 W.hr/m^2 for rotating or scanning antennas (beams).

The 1984 U.S.S.R. ELs in the frequency range 30 kHz to 300 MHz for the general population are shown in Table 10. No H-field ELs were specified. For the frequency range 300 MHz to 300 GHz, the limit on power density was 0.1 W/m^2 (0.01 mW/cm^2).

TABLE 10: U.S.S.R. STANDARD FOR PUBLIC EXPOSURE TO RFEMF (1984)
[Czerski (1985)]

Frequency (MHz)	E-Field EL (V/m)
0.03-0.3	25
0.3-3	15
3-30	10
30-300	3

An English translation of several current RFEMF-exposure standards of the Russian Federation was obtained recently [Pakhomov (1998)], a brief summary of which is given below. The original document in Russian presented no rationale for the limits stated.

2.1.8. (PHYSICAL FACTORS OF THE ENVIRONMENT) SO Sanitary Rules and Norms
[HS 2.1.8 (1996)]

These Sanitary Rules by the State Commission of Sanitary and Epidemiological Supervision of Russia became effective on May 8, 1996, and are mandatory for all government and social establishments, enterprises, and any other businesses and organizations (regardless of their form of property or affiliation), for all officials and citizens. They are also obligatory for diplomatic and other missions of foreign countries and international organizations on the territory of the Russian Federation. However, they do not apply to situations when electromagnetic radiations are intentionally used in medical practice for therapy or diagnosis. They establish separate maximum permissible exposure levels (MPELs) in the frequency range from 30 kHz to 300 GHz for occupational and general-public exposure.

For occupational exposure, MPELs are determined by a so-called "maximum exposure energy" (MEE). For the range 30 kHz to 300 MHz, the MEE is defined as a product of the square of the electric or magnetic field strength (in V/m or A/m), and the duration of exposure in hours per day. For the 300-MHz to 300-GHz range, the MEE is defined as a product of the incident power density (in $\mu W/cm^2$) and the duration of exposure in hours per day.

The MPELs are calculated by dividing the MEE limits in the frequency ranges below by the number of exposure hours per day:

30 kHz to 3 MHz: electric-field squared: 20,000 $(V/m)^2$ or magnetic-field squared: 200 $(A/m)^2$
3 MHz to 30 MHz: electric-field squared: 7,000 $(V/m)^2$ or magnetic-field squared: (not stated)
30 MHz to 50 MHz: electric-field squared: 800 $(V/m)^2$ or magnetic-field squared: 0.72 $(A/m)^2$
50 MHz to 300 MHz: electric-field squared: 800 $(V/m)^2$ or magnetic-field squared: (not stated)
300 MHz to 300 GHz: 200 $\mu W/cm^2$.

As examples, the MPEL for 8 hours of exposure per day in the 300-MHz to 300-GHz range is 200/8 = 25 $\mu W/cm^2$. For 1 hour of exposure per day in that range, the MPEL is 200/1 = 200 $\mu W/cm^2$, and for 0.2 hour of exposure per day, it is 200/0.2 = 1,000 $\mu W/cm^2$. However, if the exposure duration is less than 0.2 hour per day in the 300-MHz to 300-GHz range or less than 0.08 hour per day in the 30-kHz to 300-MHz range, no further MPEL increase is permitted.

For the general public (i.e., residential and recreation areas, inside and outside buildings, schools, hospitals, etc., and also at work places of persons under 18 and pregnant women), the Rules establish the following MPELs (which are not dependent on the exposure duration):

30 kHz to 300 kHz: 25 V/m
300 kHz to 3 MHz: 15 V/m
3 MHz to 30 MHz: 10 V/m
30 MHz to 300 MHz: 3.0 V/m
300 MHz to 300 GHz: 10 $\mu W/cm^2$.

A few exceptions and special situations are also addressed: MPELs are specified for the following TV-broadcasting frequencies: 48.4, 88.4, 192, and 300 MHz, for which the respective MPELs are 5, 4, 3, and 2.5 V/m. The exposure levels from "special-purpose" radars (which are designed for outer-space location and operate in a beam-scanning mode in a 150-300 MHz band) are set at 10 $\mu W/cm^2$ (6 V/m) in the near-field zone and at 100 $\mu W/cm^2$ in the far field. Also, the MPEL in the 300-MHz to 300-GHz band is increased to 100 $\mu W/cm^2$ for exposure from rotating and beam-scanning operation antennas (less than 60 r.p.m. and more than a duty ratio of 20).

There are also special rules for the city of Moscow to protect its population from the fields emitted from "radiofrequency transmitting objects" (RTO), to take into account the large number and concentration of such objects in the city. These rules for Moscow can be overridden by federal rules in case the latter dictate stricter criteria for protection from such fields. The definition "RTO" applies to any radiofrequency transmission means, including radio and TV broadcasting centers, radiolocation and relay stations, ground-based satellite communication systems, and military equipment.

Also specified are permissible safety levels for various home appliances and for people living near cellular-telephone base stations and for users of cellular telephones, rules that are applicable to the Republic of Byelorussia [Belarus] as well as the Russian Federation.

The more-stringent U.S.S.R. standards than those of the U.S.A. reflected basic differences in philosophy in the standard-setting processes of the two countries. In the U.S.A., small deviations from physiological norms are not necessarily regarded as clinically significant, thereby recognizing that an effect may be real, but may not be a hazard. By contrast, Trakhtenberg in the U.S.S.R. (quoted in Goldmann, 1982) had defined significant physiological changes as "characterized by the deviation of the factors studied beyond the limits of annual or seasonal fluctuations by more than two standard deviations away from the norm." In many cases, however, such deviations from norms may not necessarily have medically important implications. In the absence of the rationale in the current Russian Federation standards (or the copy in Russian obtained), presumably the standard-setting processes used in the Russian Federation are based on similar differences in philosophy.

2 INTERACTIONS OF RFEMF WITH BIOLOGICAL ENTITIES

2.1 RFEMF VERSUS IONIZING RADIATION

Some non-specialists may not know about the basic differences between "nonionizing" electromagnetic fields (having frequencies fron 0 to 300 GHz) and "ionizing" radiation (having frequencies many orders of magnitude higher than for RFEMF or at the correspondingly shorter wavelengths). They therefore may be concerned that the well-known hazards of exposure to ionizing radiation can also occur from exposure to RFEMF.

Physics teaches that all forms of electromagnetic energy propagate as streams of individual energy packets (each called "a quantum"), and that the amount of energy in any quantum is directly dependent on its frequency, i.e., the higher the frequency of a quantum, the greater its intrinsic energy. Ionizing radiations, such as ultraviolet light, X-rays, the emissions from radioactive materials, and gamma and cosmic rays have frequencies ranging from millions to trillions of times higher than those of RFEMF, and therefore have correspondingly higher energies. Moreover, a single quantum of any of those radiations has enough intrinsic energy to ionize (eject an electron from) a molecule, hence the term "ionizing". The ejection of an electron from a biological molecule leaves the molecule positively charged, thereby greatly altering its own properties and enhancing its electrical and biological interactions with its neighboring molecules. The resulting effects on biological tissues that absorb such quanta can be cumulative and irreversible, and for humans, can profoundly affect their health.

Members of the public are exposed in varying degrees to naturally occurring forms of ionizing radiation, including ultraviolet light from the sun, radioactive materials in the earth (including those released from mining and burning of coal and the release of radon gas), and gamma and cosmic rays from outer space. A well-known example of a hazard from ionizing radiation is overexposure to the ultraviolet rays from the sun, which can cause skin cancer. Added to the natural emissions are those from human-created devices such as X-ray machines for various purposes (diagnosis, dentistry) and ultraviolet lamps for sun tanning.

By contrast, because of the vastly lower frequencies of nonionizing radiations such as RFEMF, their quanta have intrinsic energies far too small (by factors of at least 100,000) to ionize molecules in a biological entity. However, they can agitate the molecules when arriving in large numbers per unit of time and thereby add heat to the entity. At low arrival rates, the increase in molecular agitation is small, and the added heat can be dissipated readily by various existing heat-removal mechanisms. Thus, RFEMF absorption as heat within such an entity would not be biologically significant unless the quantum arrival rate is high enough to exceed such dissipation mechanisms.

The heat produced within mammals of a given species by exposure to RFEMF at relatively low incident power densities normally can be compensated for by the thermoregulatory capabilities of that species, so if any thermal effects are produced, they are usually reversible. Moreover, the molecular agitation or heat added by the RFEMF is no longer produced when exposure ceases, and the heat generated before the cessation of exposure is dissipated quickly, so the quantities of heat induced by successive RFEMF exposures at such levels would not be cumulative. At relatively high intensities, however, the heat produced may exceed the thermoregulatory capabilities of that species, so physiological compensation for such heating may be inadequate. Thus (depending on the species), RFEMF exposure at high intensities (quantum arrival rates) could cause thermal distress or even irreversible thermal damage.

The general public is exposed to electromagnetic fields from many kinds of generators and emitters designed by humans. More than a decade ago, the EPA had measured the environmental field intensities at selected locations in 15 U.S. cities [Tell and Mantiply (1980), Janes et al. (1977), Hankin (1985), EPA (1986a)]. The median exposures at that time ranged from a low of 0.002 $\mu W/cm^2$ (for San Francisco and Chicago) to a high of 0.020 $\mu W/cm^2$ (for Portland, Oregon); the population-weighted median for all 15 cities was 0.0048 $\mu W/cm^2$. The major sources of those exposure values were from FM-radio and TV broadcast stations. Other sources included the various types of radar and communications

systems (both public and private). Also reported were the emission levels in the vicinity of specific transmitting antennas, some of which exceeded the then current exposure guidelines [EPA (1986b), Tell and O'Brien (1977)].

A continuing need is to assess whether any deleterious effects on human health would result from the increasing proliferation of systems that intentionally broadcast electric and/or magnetic fields, as well as the fields emitted by home appliances (such as microwave ovens) and the increasing use of new technologies. Included in the latter is the explosion in wireless telecommunication devices dsuch as cellular and PCS phones, pagers, and so forth. Since those EPA measurements had been made, the median levels certainly increased by large factors, but would have had to rise by more than about 100,000 times to exceed maximum allowable exposure levels prescribed in current guidelines.

Recently, Mantiply et al. (1997) summarized the measured data on the electric and magnetic fields in the frequency range 10 kHz to 30 GHz, derived from various environmental and occupational sources, and cited the literature from which those data were derived. Table 1 of the paper is a list of the standard frequency-band designations (VLF etc.) and sources of such emissions. Figures 1-8 of the paper are graphs that display electric-field strengths and magnetic-field strengths as log ordinates versus wavelengths and frequencies as linear abscissas for each standard band, with shaded areas to indicate the sources and distances therefrom or specific locales where the measurements were done. The overlapping areas are cross-hatched and indicate a range of field values with ratios of E to H close to 377 ohms (the impedance of free-space). As a caveat in near-field cases, the overlap may be coincidental and not related to the impedance of free-space.

Presented in the text are more complete data for each band than shown in the graphs, including considerations of time variations (or modulation), peak-versus-average values, and spatial character of the fields. Subheadings for each band that describe specific types of transmitters that emit energy in that band as follows:

<u>VLF (Very Low Frequency: 10 to 30 kHz)</u>
Omega Navigational Transmitters
VLF Submarine Communications System
Visual Display Terminals
Induction Heating Stoves
LF (Low Frequency: 30 to 300 kHz)
Loran Navigational Transmitters

<u>MF (Medium Frequency: 300 kHz to 3 MHz)</u>
AM Standard Broadcast
Amateur Radio (160 Meter Band)
Induction Heaters
Electrosurgical Units

<u>HF (High Frequency: 3 to 30 MHz)</u>
Amateur Radio
Citizens Band Radio
International Broadcast
Dielectric Heaters
Shortwave Diathermy

<u>VHF (Very High Frequency: 30 to 300 MHz)</u>
FM Radio Broadcast
VHF Television Transmitters
Mobile Transmitters
Portable Transmitters

UHF (Ultra-High Frequency: 300 MHz to 3 GHz)
UHF Television Transmitters
Cellular Telephones
Microwave Ovens
Microwave Diathermy
Pulsed Radar

SHF (Super-High Frequency: 3 to 30 GHz)
Microwave Relay
Satellite Communications Uplinks
Aircraft Onboard Weather Radars
Police Traffic Radar

In the abstract of the paper, the authors stated that for the sources included: "The strongest fields are found near industrial induction and dielectric heaters, and close to the radiating elements or transmitter leads of high power antenna systems. Handheld transmitters can produce near fields of about 500 V/m at the antenna. Fields in the general urban environment are principally associated with radio and TV broadcast services and measure about 0.1 V/m root-mean-square (rms). Peak fields from air traffic radars sampled in one urban environment were about 10 V/m, 300 times greater than the rms value of 0.03 V/m when the duty factor associated with antenna rotation and pulsing are factored in."

2.2 EFFECTS VERSUS HAZARDS

An effect produced in a body by exposure to electromagnetic radiations may not necessarily be harmful to that body. As an example of a nonhazardous effect, the absorption of visible light [electromagnetic radiation having intrinsic (quantum) energies far above those of RFEMF but well below those of ultraviolet light and the other ionizing radiations mentioned previously] in the eyes is essential for vision. However, light at very high intensities (arrival of many quanta per second) may damage the retina. Also noteworthy is that visible and infrared radiation are largely absorbed in the skin, and at normal levels are converted into harmless heat, but as indicated previously, excessive absorption of ultraviolet light can cause skin cancer.

2.3 THERMAL INTERACTIONS AND SPECIFIC ABSORPTION RATES (SARs)

The bodies and tissues of most plant and animal species contain little if any intrinsic magnetic material, so the interactions of RFEMF with a biological body largely depend on the electrical and thermal properties of the body's various constituents and the distribution of those constituents within that body. Such interactions are also dependent on the characteristics of the incident RFEMF [its frequency (intrinsic energy), power density (intensity), and polarization (relative to the body's orientation)].

Although most bodies are not very transparent to visible light, their interactions with RFEMF obey the optical laws of reflection and refraction at their surfaces. Because the index of refraction of any material is related to its dielectric properties, RFEMF incident on a body is reflected and refracted at the boundaries between regions of significantly different dielectric properties, such as at the body's air-surface interface. This is true for the same physical reasons as those that apply to light incident at an air-glass interface, and is also applicable at the internal boundaries between different constituents of the body, thereby affecting the spatial variation of electric field with internal location. Figure 1 is a graph of the fraction of power transmitted (not reflected) at an air-muscle interface versus RFEMF frequency. At 3.0 GHz, for example, about 44% of the incident power density enters the body, with the remaining 56% reflected at the surface.

The fraction of incident power density that enters a body undergoes progressive attenuation with depth because of energy absorption. The term "penetration depth" is usually used to quantify such attenuation. For objects with homogeneous properties and with the RFEMF incident at right angles to the surface, the penetration depth is defined as the distance at which the power density is decreased by absorption to about 14% of its value just within the body's surface. Graphs of penetration depth versus

frequency for muscle, blood, and fat are shown in Figure 2. It is seen that the penetration depth becomes smaller as the frequency of the RFEMF is raised. At 3.0 GHz, for example, the penetration depth for muscle and other tissues having a high water content is about 1.7 cm (2/3 of an inch). Not shown in that figure is that penetration depth at about 30 GHz and higher is largely confined to the outer layers of the skin (much like for sunlight).

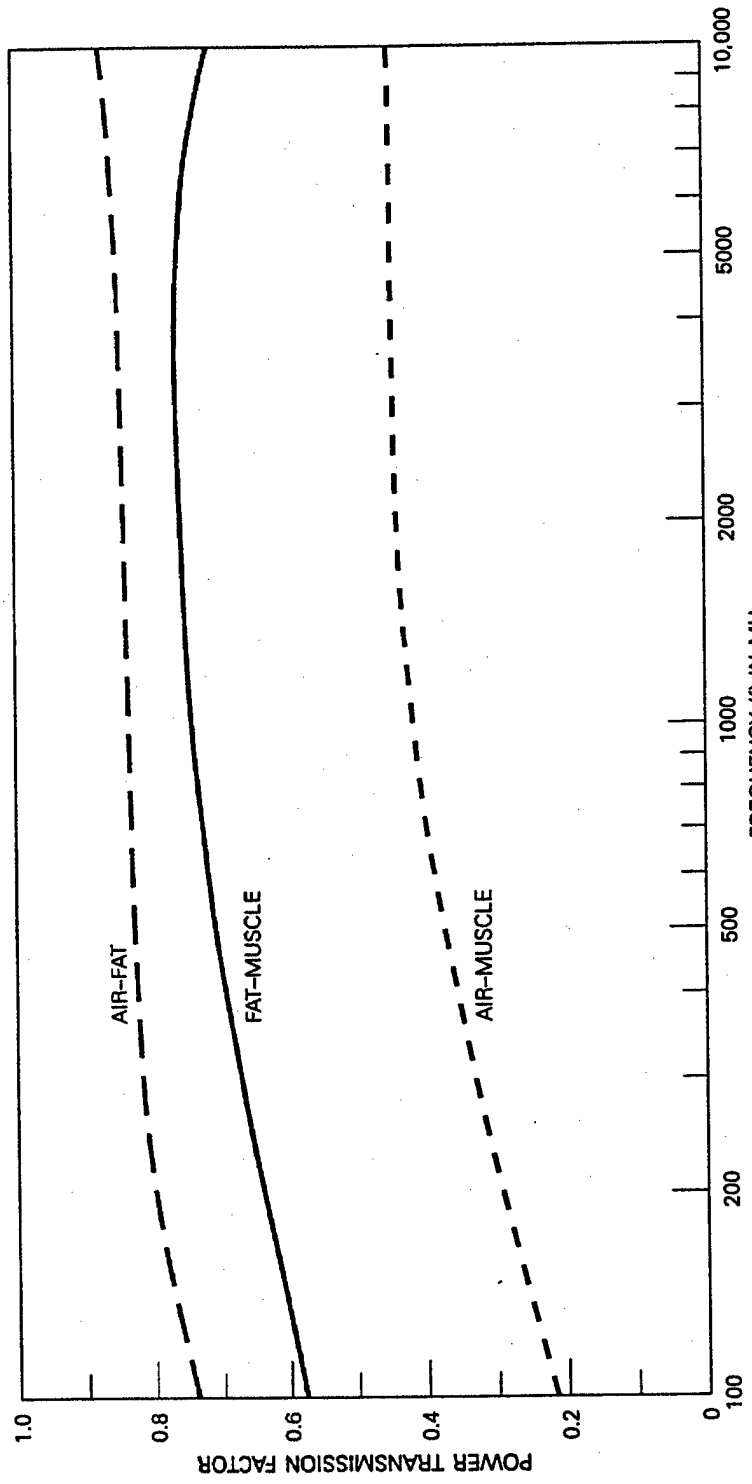

FIGURE 1 POWER TRANSMISSION FACTORS FOR AIR, FAT, AND MUSCLE INTERFACES

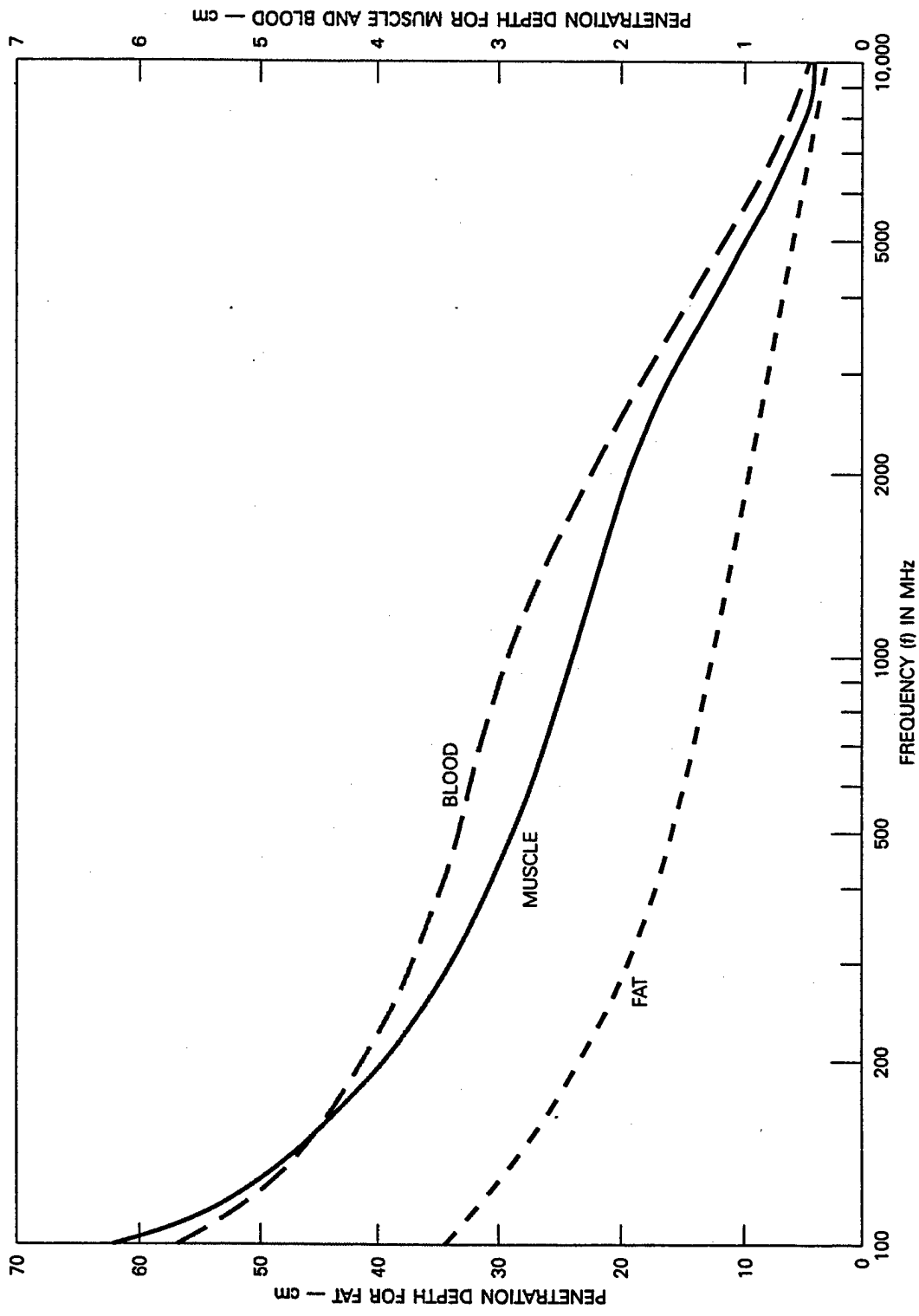

FIGURE 2 PENETRATION DEPTH VERSUS FREQUENCY FOR MUSCLE, BLOOD, AND FAT

In publications on the bioeffects of RFEMF, the rate of energy absorbed by the constituents within any small volume of a body (nominally 1 cubic centimeter) from an incident electromagnetic field is usually termed the "local specific absorption rate" [or "local SAR"]. Its values are expressed in watts per kilogram (W/kg) of the mass in that volume. The local SAR thus defined at any site within a body depends on the characteristics of the incident RFEMF (carrier frequency, modulation, amplitudes and directions of its components), and on the location of the volume and its dielectric and thermal properties. For bodies of complex shape and large internal spatial variations in properties, local SAR values are difficult to determine by experiment or calculation. Instead, the spatial average SAR for the body, called the "whole-body SAR" (or often simply the SAR), is frequently used because it can be measured without requiring any information on the internal variations of the local SAR.

In early investigations, whole-body SARs were calculated for models of relatively simple geometry such as spheroids, ellipsoids, and cylinders that have weights and dimensions approximately representative of various species, including humans. Such calculations had been experimentally verified by exposing physical models in various orientations to linearly-polarized plane-wave RFEMF and determining distributions of the heat produced therein.

A general finding is that the largest value of whole-body SAR of a human or animal figure exposed to linearly-polarized plane-wave RFEMF would occur if the figure is in the "E" orientation [its longest body axis parallel to the electric field] and if the RFEMF wavelength is about 2.5 times the length of the body axis, or conversely, if the length of that axis is about 0.4 of the corresponding wavelength). The adjective "resonant" is used for that wavelength and its corresponding frequency. At resonance, the model absorbs RFEMF energy much like a lossy half-wave dipole at its resonant frequency. Exposures at other orientations experimental investigations yield lower SARs.

Many of the important results of such theoretical and experimental studies were embodied in a series of handbooks issued by the U.S. Air Force. The last handbook issued (Durney et al., 1986) includes the information in the previous editions and contains much other pertinent information as well. This edition was made available as a compact disk from the Air Force Research Laboratory at Brooks Air Force Base, TX, or on the internet at: http://www.brooks.af.mil/AL/OE/OER/handbook/cover.htm.

Of continuing interest are the plots of calculated whole-body SAR versus frequency for prolate-spheroidal models of an "average" man, woman, and 5-year-old child for exposure to 1 mW/cm^2 in three orientations (pp. 6.4, 6.7, 6.9 of the printed version), reproduced herein as Figures 3, 4, and 5. Analogous plots for a prolate-spheroidal model of a medium rat (p. 6.17) are shown in Figure 6 for comparison. Those plots all display the aforementioned resonances in the E-orientation, with sharp reductions in SAR below each resonant frequency and slower decreases above that resonant frequency.

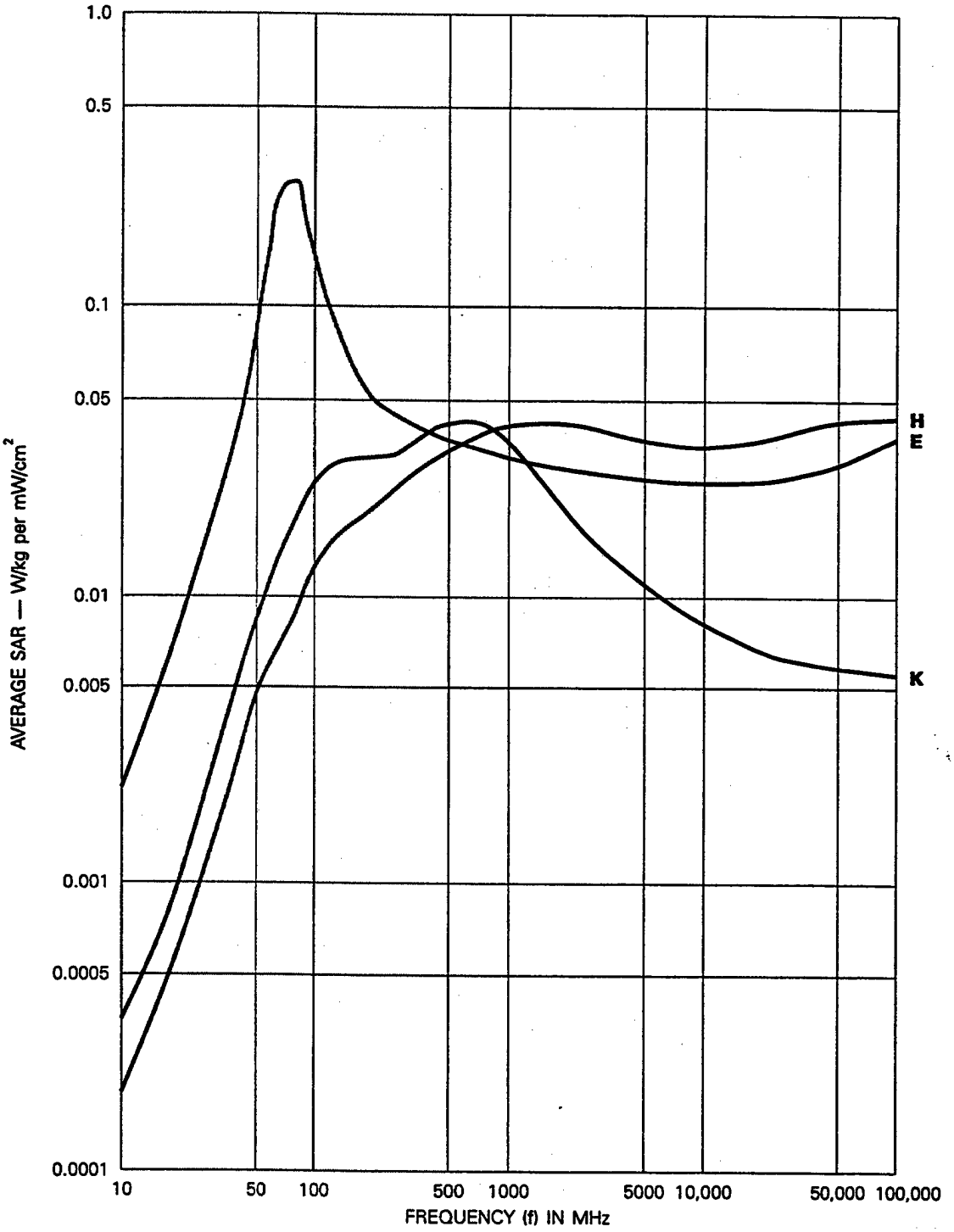

FIGURE 3 CALCULATED PLANE-WAVE AVERAGE SAR VERSUS FREQUENCY FOR AN AVERAGE MAN IN THE E, H, K ORIENTATIONS (Prolate spheroidal model, three polarizations; a = 0.875 m, b = 0.138 m, V = 0.07 m^3.)

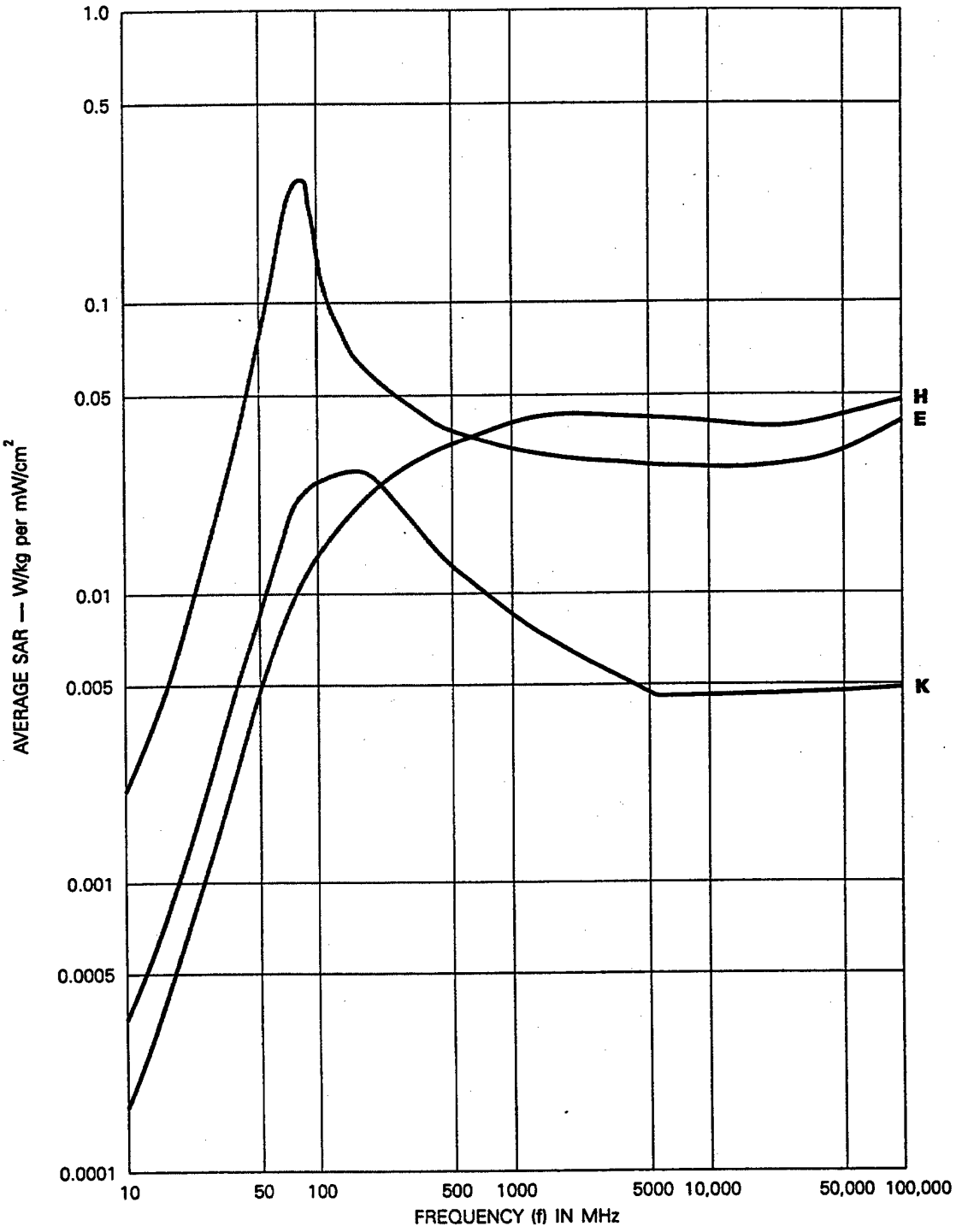

FIGURE 4 CALCULATED PLANE-WAVE AVERAGE SAR VERSUS FREQUENCY FOR AN AVERAGE WOMAN IN THE E, H, K ORIENTATIONS (Prolate spheroidal model, three polarizations; a = 0.805 m, b = 0.135 m, V = 0.06114 m^3.)

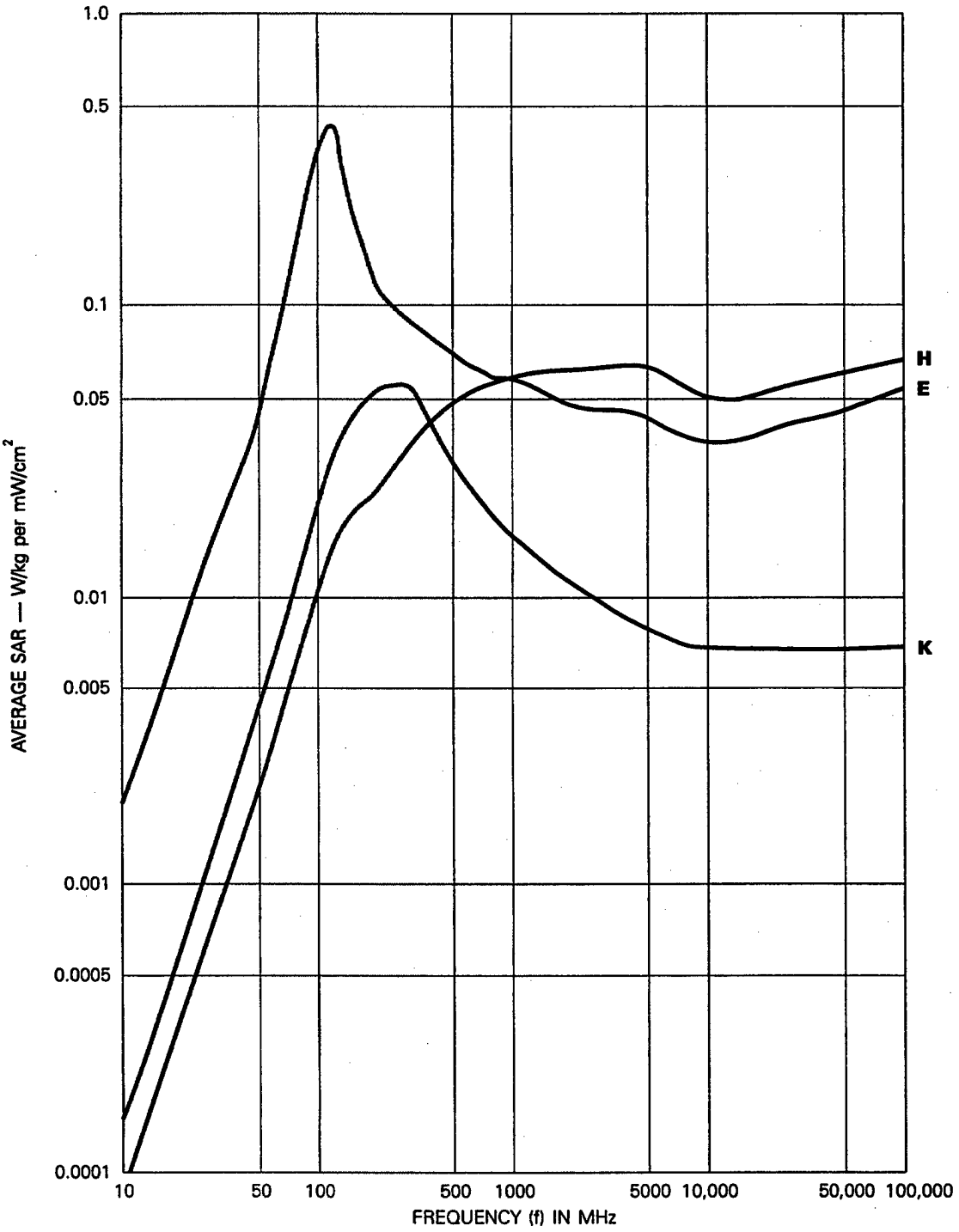

FIGURE 5 CALCULATED PLANE-WAVE AVERAGE SAR VERSUS FREQUENCY FOR AN AVERAGE 5-YEAR-OLD CHILD IN THE E, H, K ORIENTATIONS (Prolate spheroidal model, three polarizations; a = 0.56 m, b = 0.091 m, V = 0.0195 m^3.)

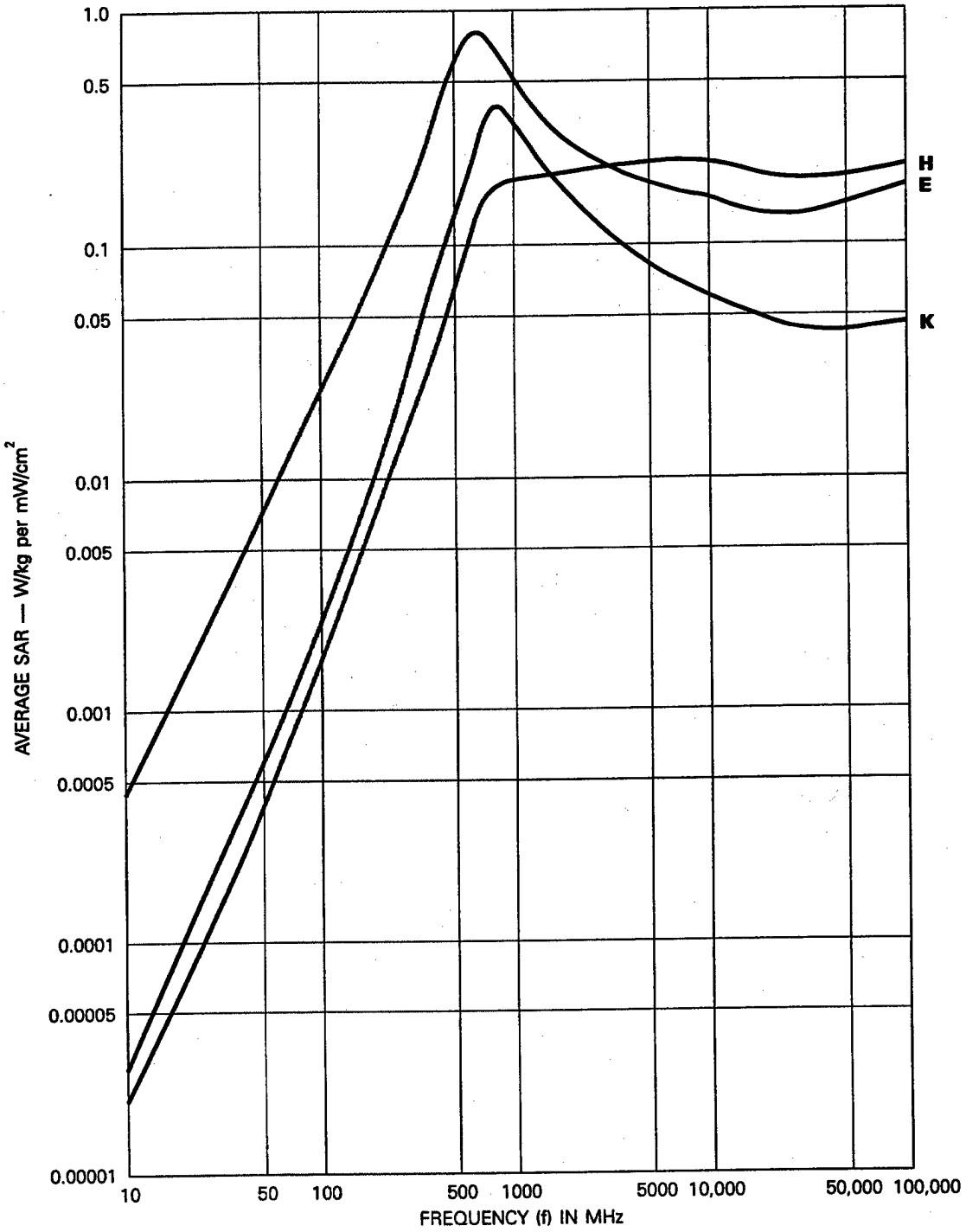

FIGURE 6 CALCULATED PLANE-WAVE AVERAGE SAR VERSUS FREQUENCY FOR A MEDIUM RAT IN THE E, H, K ORIENTATIONS
(Prolate spheroidal model, three polarizations; a = 0.1 m, b = 0.0276 m, V = 3.2×10^{-4} m^3.)

Specifically, the resonant frequency for the "average" man, taken to be 5 feet 9 inches tall (1.75 m) and weighing about 154 lb (70 kg), is about 70 MHz (when insulated from ground). At this frequency, the whole-body SAR is about 0.2 W/kg for an incident plane-wave power density of 1 mW/cm^2. This SAR is about 1/6 of his resting metabolic rate or 1/20 to 1/90 of his metabolic rate when doing exercise ranging from walking to sprinting. By calculation, exposure of a man at this SAR (to 1 mW/cm^2) for, say, 1 hour would produce a mean temperature increase of about 0.2°C if no mechanisms for heat removal (conduction, convection, or radiation) were operating. The actual temperature increases would be smaller with such heat-exchange mechanisms present. In addition, the compensation exercised by the thermoregulatory systems of live mammals may prevent any rises in body temperature.

Similarly, the resonant frequency for a prolate-spheroidal model of an "average" woman about 5 feet 3 inches tall (insulated from ground), is about 80 MHz, and her mean SAR is about the same as for the average model man. For the model of a 5-year-old child, the resonant frequency is about 110 MHz, and the resonant SAR is about 0.3 W/kg per mW/cm^2.

By contrast, the resonant frequency for a prolate-spheroidal model of a medium rat is about 650 MHz, and the resonant whole-body SAR is about 0.8 W/kg per mW/cm^2. These values and those for other laboratory animals in RFEMF-bioeffects studies are important in endeavoring to relate the results of such animal studies to possible effects in humans.

Calculations indicate that if humans were to stand in bare feet on a wet surface, their resonant frequencies would be approximately halved but their whole-body SARs (at the lower resonant frequency) would be higher.

Under some conditions, the SARs of various parts of the body, such as the head and limbs, also require consideration. In an important early study, Shapiro et al. (1971) calculated distributions of fields that would be induced in a multilayered spherical model of a primate head from exposure to 3.0-GHz plane-wave RFEMF. Their calculations indicated the existence of local internal regions of relatively high electric fields. Johnson and Guy (1972) obtained experimental results that confirmed the presence of such regions. Kritikos and Schwan (1972, 1975) did similar studies for frequencies in the range from 300 MHz to 12 GHz. In general, the locations of such regions depend on the size of the head, the electromagnetic and thermal properties of its layers, and the frequency of the incident field. Such regions have been dubbed "hot spots", even for combinations of power density and exposure duration that would yield biologically insignificant temperature rises at such spots.

Numerical calculations of internal spatial distributions of SAR have been done for "block" models. In such models, the shape of a body is approximated by an appropriate arrangement of many rectangular cells or blocks of various sizes, with each block assumed to be biologically homogeneous and to have constant internal field over its volume when the model is exposed to RFEMF. Also, the biological properties ascribed to each block are selected to approximate those of the tissues in the corresponding location of the body. By spatial averaging over such models, more accurate values of whole-body SAR have been derived than from simpler ones.

Rukspollmuang and Chen (1979), using a block model of an isolated multilayered spherical head, obtained results that were qualitatively similar to those of Kritikos and Schwan (1975). They then studied, at 918 MHz and 2.45 GHz, a block model with a shape and an internal structure more closely approximating that of the human head (including eyes, nose, skull bone, and brain), and found that much of the energy would be absorbed within the skull. Also, frontal exposure of the model to 2.45 GHz would induce fields that are primarily concentrated near the front surface, so energy dissipation within the brain would be relatively low.

Hagmann et al. (1979) calculated SAR distributions in the attached head of a block model of a human, and derived whole-head and whole-body SARs for three orientations of the model relative to the source of RFEMF. For front-to-back propagation with the long axis of the body parallel to the electric vector, they found a broad head resonance at about 350 MHz, with a whole-head SAR of about 0.12 W/kg per mW/cm^2; the corresponding whole-body SAR was about 0.05 W/kg per mW/cm^2. For RFEMF

propagation in the head-to-toe direction, a sharper head resonance at 375 MHz was obtained, with whole-head and whole-body SARs respectively approximately 0.22 and 0.07 W/kg per mW/cm^2. Many results of such numerical analyses of whole-body SARs and internal SAR distributions were experimentally verified.

A number of researchers [including Gandhi et al. (1992a, 1992b), Olsen (1982), Olsen and Griner (1987, 1989, 1993) and others] studied figurines of humans and animals fabricated from synthetic biological materials having electromagnetic characteristics that approximated their various biological constituents. In some of those studies, such figurines were exposed to RFEMF at power densities sufficient to produce accurately measurable temperature increases, and such temperature rises were determined immediately after exposure. An important early qualitative result is that at frequencies near resonance, local internal fields for human figurines can be much higher for regions such as the neck and groin than for other locations in the body. Also, for nonprimate figurines, the variations of internal field with location are quite different from those for primate figurines, findings that must be considered when endeavoring to relate experimental results for laboratory animals to humans, or when comparing experimental results between two (or more) laboratory species.

More recently, SAR distributions within human figurines or specific regions thereof have been determined with computer algorithms such as the Finite-Difference-Time-Domain (FDTD) method and others. A RFEMF-dosimetry workshop covering this and other topics was held in 1996 at Brooks Air Force Base, Texas, and a workshop report (Hurt, 1996) was issued.

Under similar exposure conditions, the whole-body SARs obtained with amplitude-modulated (AM) RFEMF at any given carrier frequency and average power density are the same as those of CW RFEMF or frequency-modulated (FM-CW) RFEMF. In the context of nonthermal interactions, discussed below, it should be noted that the term "local SAR" denotes the rate of energy absorption at any local site within a biological object, and does not necessarily indicate that such absorption occurs as heat. Rather, it is a useful measure of the local field strength resulting from RFEMF exposure, especially at internal field strengths too low to produce heat at biologically significant rates.

A compendium of the dielectric properties of various body tissues was produced by Gabriel and Gabriel (1996) for the Air Force Research Laboratory at Brooks Air Force Base in Texas. Like the previously mentioned handbook (Durney et al., 1986), this compendium is available on the internet at http://www.brooks.af.mil/AL/OE/OER/dielectric/cover.htm.

2.4 NONTHERMAL INTERACTIONS AND SARs

RFEMF pulses having specific characteristics can be perceived by some humans as apparent sound (the "RFEMF-auditory effect"). Because each pulse in a train of pulses can be perceived individually if the pulses are widely spaced in time (with correspondingly very low time-averaged SARs), this effect has been characterized by some investigators as nonthermal. Pulsed RFEMF has also been reported by some researchers to produce other effects in animals, such as alterations of the blood-brain barrier and behavioral changes. Still other researchers who used RFEMF that is amplitude-modulated at specific frequencies (mostly below about 30 Hz but up to about 400 Hz), have reported biological effects from the amplitude modulation per se, notably the "calcium-efflux" effect.

Reports on such findings are regarded by some investigators as evidence for the occurrence of nonthermal RFEMF bioeffects, but others dispute the existence of those effects, a subject that is not likely to be resolved in the near future.

2.5 DISTINCTION BETWEEN RFEMF AND FIELDS AT POWERLINE FREQUENCIES

Worth mentioning under the heading "nonionizing fields" are several forms of electric and magnetic fields. Such fields occur naturally, including the earth's magnetic field and the electric fields in the atmosphere (most prominent during storms), and artificially in systems developed by humans for

generating them for various uses. Much controversy exists about reports of deleterious effects on humans from exposure to the electric and magnetic fields in homes at frequencies much below those of RFEMF (such as those from appliances in operation), from the power lines supplying the electricity, and the fields from any nearby high-tension power lines. In considering possible bioeffects of the RFEMF from any specific system, it is important to recognize the distinction between such powerline fields and the fields emitted (at much higher frequencies) from such RFEMF systems.

Most powerline sources in the U.S. operate at 60 Hz. The corresponding wavelength is more than 3,000 miles, meaning that people near a powerline are in its "induction zone", within which "propagation", "radiation", and similar terms do not apply. Rather, electric and magnetic fields from such sources can cause or induce currents in the body that alternately flow in the positive and negative directions once during each cycle at the 60-Hz rate, and the effects of each field should be considered separately.

At 3.0 GHz, for example, which is 50 million times higher than 60 Hz, the corresponding wavelength is only about 4 inches. Thus, the antennas of sources operating at such high frequencies emit and propagate electromagnetic fields as true radiation, even at short distances from the antennas. In such radiation, the electric and magnetic components are normally at right angles to one another and to the directions of propagation. In addition, the ratio of the intensity of the electric component to that of the magnetic component has a constant numerical value, so the intensity of the RFEMF can be stated in terms of the intensity of either component alone.

Based partly on the foregoing, a major difference between powerline and microwave fields is the way in which each interacts with a biological body with regard to penetration and absorption. In any small region within the body, each powerline field adds to and subtracts from, at the 60-Hz rate, the internal fields present in that region in the absence of the external fields. A 60-Hz electric field is attenuated internally by 100,000 to a million times by the dielectric properties of the body's constituents at that frequency. On the other hand, a 60-Hz magnetic field pervades the body with little if any change, so it is appropriate to consider the currents induced in the body by such magnetic fields. Whether electric or magnetic fields at such frequencies can cause biologically significant effects depends largely on how much they alter the body's intrinsic electric fields or currents.

Especially alarming to the general public, based mostly on a number of epidemiologic and animal studies, is the reported association between exposure to 60-Hz or 50-Hz magnetic fields and the incidence of one form or another of cancer. It should be noted, however, that finding a statistically significant association in epidemiologic studies does not necessarily indicate the existence of a causal relationship, and the positive findings of some experimental studies with laboratory animals are questionable, mostly because the experiments performed were flawed. In the view of various analysts, including those who prepared the NAS (1996) report, the findings at powerline frequencies are not strong enough to warrant a positive statement that such effects are real, but that the possible existence of such effects cannot be dismissed either. In any case, the interaction mechanisms at powerline frequencies are quite different from those at RFEMF frequencies and any positive findings of either would likely not apply to the other.

3 RFEMF AND CANCER IN HUMANS

Much of the information regarding whether there exists an association between cancer incidence in humans and exposure to RFEMF is derived from epidemiologic studies, discussed in Section 3.1 below. There are cases in which individuals have claimed that they or one of their relatives had developed one form or another of cancer from being exposed to RFEMF. In some of those cases, legal proceedings were instituted, with various outcomes. Several such cases are discussed in Section 3.2.

In experimental studies with non-human species, well designed protocols can yield a good measure of control over experimental errors, and possible confounding factors usually can be diminished to a far greater extent than in epidemiologic studies. Thus, much of the evidence for the existence of an association between RFEMF and cancer incidence or for the absence of such an association is based on the findings of experimental studies. However, such findings comprise only indirect evidence for or

against similar effects in humans because of the large differences among species and the consequent problems of extrapolating findings of such experimental studies to humans. Such experimental studies are discussed in Section 4.

3.1 EPIDEMIOLOGIC/OCCUPATIONAL STUDIES

Robinette and Silverman (1977), in a study of males (mostly white) who had served in the Navy during the Korean War, selected 19,965 equipment-repair men as having had occupational exposure to RFEMF based on their titles of Electronics Technician, Fire Control Technician, or Aircraft Electronics Technician. For comparison, they selected 20,726 Naval equipment-operation men who by their titles of Radioman, Radarman, or Aircraft Electrician's Mate presumably had little occupational exposure to RFEMF, and for brevity denoted them as the "control group". The mean age of the control group was about 1.5 years lower than for the exposed group. Studied were extant mortality records for 1955-1974, in-service morbidity for 1950-1959, morbidity for 1963-1976 in Veterans Administration hospitals, and records of both granted and disallowed requests in 1976 for disability compensation.

Only the mortality results were presented in this paper. There were 619 deaths (3.1%) from all causes in the exposed group versus 579 deaths (2.8%) in the control group; the difference was statistically nonsignificant. The death rates of both groups were lower than for comparable age-specific white males (937 and 916) in the U.S. population at large.

Those decedent data showed no significant difference between exposed and control groups in deaths from all diseases, respectively 311 (1.6%) and 321 (1.5%), both significantly lower than for corresponding groups in the age-specific general white-male population. The death rate from trauma was significantly higher ($p<0.01$) in the exposed than the control group, 295 (1.5%) versus 247 (1.2%). However, when the trauma deaths were divided into accidents (motor-vehicle and "other"), suicides, and homicides, the only significant difference ($p<0.01$) between exposed and control groups was in the "other-accident" category, 130 (0.65%) versus 70 (0.34%). Examination of the death certificates and other mortality information about those in the exposed group showed that many of those men had died in military-aircraft accidents after the Korean War (5.3% versus 2.3%), presumably because more of them later became flying officers.

The deaths from disease were divided into the following categories: all malignant neoplasms; cardiovascular [including vascular lesions of the central nervous system (strokes), and arteriosclerotic heart]; chronic nephritis, other renal; influenza and pneumonia; and cirrhosis of the liver. In all of those categories, the totals were less than those in the U.S. age-specific white male population. Also, none of the differences in total numbers between the exposed and control groups were significant, but the numbers of deaths specifically associated with arteriosclerotic heart disease were 8 (0.04%) versus 26 (0.13%), significantly lower ($p<0.05$) in the exposed group than the control group.

The authors tabulated the cancer deaths separately, as shown in Table 11 (adapted from their Table 7). There were 96 deaths from "All Malignant Neoplasms" among those exposed occupationally versus 127.4 expected deaths (a ratio of 0.75). There were also 84 deaths versus 119.1 expected deaths among the controls (a ratio of 0.71), which yielded a risk ratio RR (ratio of ratios) of 1.07 (non-significant, $p>0.05$). Also, none of the RRs for specific cancer deaths were significant.

TABLE 11: CANCER MORTALITY IN U.S. VETERANS
[Robinette and Silverman (1977)]

ICD 8th Rev. Code	Cause	Exposed Recorded	Exposed Expected	Controls Recorded	Controls Expected	Risk	P*
140-209	All Malign. Neoplasms	96	127.4	84	119.1	1.07	NS
140-149	Buccal Cavity & Pharynx	2	2.8	2	2.5	0.89	NS
150-159	Digestive Organs	20	24.1	12	21.6	1.49	NS
160-163	Respiratory Tract	25	31.0	17	26.9	1.28	NS
180-187	Genital Organs	5	6.6	5	7.7	1.06	NS
188-189	Urinary Organs	2	2.4	4	1.4	0.29	NS
204-207	Leukemia & Aleukemia	12	11.2	7	11.3	1.72	NS
200-203	Other Lymphatic	12	18.2	12	18.3	1.00	NS
208-209	Other	18	29.2	25	28.2	0.70	NS

*NS: P>0.05

Silverman (1979), in a review of RFEMF-epidemiologic studies, included the study above of Naval personnel (with the two groups called "high-exposure" and "low-exposure" and with 144 and 55 additional men respectively in the two groups). The author, citing a paper by Glaser and Heimer, (1971)", stated: "There have been enough accidental exposures at estimated levels exceeding 100 mW/cm^2 to indicate that there are occupations in which some men at some times on certain classes of ships have been exposed well in excess of the [then] 10 mW/cm^2 limit." She also noted: "Shipboard monitoring programs in the Navy since 1957 show that men in other occupations rarely, if ever, were exposed to doses in excess of this limit. Radiomen and radar operators (our low-exposure group), whose duties keep them far from radar pulse generators and antennae, were generally exposed to levels well below 1 mW/cm^2, whereas gunfire control technicians and electronics technicians (our high-exposure group) were exposed to higher levels in the course of their duties."

The author, in addition to assessing the occupations, used individual personnel records to determine length of time in occupation, class of ship, and power of equipment on the ship at the time of exposure. Because of cost and time considerations, these records were used only for those in the high-exposure occupations who had died from nonaccidental causes and for a randomly selected 5% sample of living men in the same occupations. An index of potential RFEMF exposure called the "Hazard Number" was constructed from the information for each man. It was defined as the number of months of the man's assignment to a ship or aircraft multiplied by the sum of the power ratings of all of the gunfire-control radars aboard that ship or by all the search radars aboard that aircraft.

The percentages of men with large values of Hazard Number were much higher for the occcupations Fire Control Technician and Aircraft Electronics Technician than for Electronics Technician. However, the author did not show any comparisons of mortality data among them.

Silverman (1979) concluded that: "Differential health risks associated with potential occupational exposure to radar in the Navy more than 20 years ago are not apparent with respect to long-term mortality patterns or hospitalized illness around the period of exposure, two endpoints for which there is virtually complete information for the total study group. Later hospitalization (in Veterans Administration facilities only) and awards for service-connected disability, the two other endpoints examined, provide incomplete information. While some significant differences among the occupational groups classified by

level of potential exposure have been found with respect to all endpoints studied, the differences could not be interpreted as a direct result of microwave exposure."

Morbidity data and other health-related aspects were not presented in either paper, but Silverman (1979) indicated the possibility that effects involving the cardiovascular, endocrine, and central nervous systems may be transient and may disappear shortly after termination of exposure or not produce symptoms that warrant hospitalization.

The information above was also presented in Robinette et al. (1980). In addition, the numbers of admissions to Naval hospitals during the years 1952-1959 (except for 1955, for which the files were not available) and admission rates (per 1000 per year) for men in the low-exposure and high-exposure groups were tabulated by diagnosis (per International Classification of Diseases). Of 18 comparisons between groups, only two were significant: the low-exposure group had larger admission rates for mental disease ($p<0.001$) and larger rates for accidents, poisonings, and violence ($p<0.01$) than the high-exposure group. For none of the disease classes was the admission rate of the high-exposure group significantly larger than for the low-exposure group.

Data were also obtained for the numbers of admissions and the admission rates to Veterans Administration (VA) hospitals during 1963-1976, but it was evident that admissions to VA hospitals comprised only small fractions of hospital care of these veterans, which precluded drawing any firm conclusions. Also examined were the rates (per thousand), by diagnosis and exposure class, of men who had received VA compensation in December 1976. The only significant difference was for mental conditions, 7.1 per thousand for the low-exposure group versus 4.8 per thousand for the high-exposure group ($p<0.01$).

In a letter, Morton (1981) questioned the basis used by Robinette et al. (1980) for selecting the high-exposure and low-exposure groups, stating: "Is it possible that no suitable controls existed among personnel assigned to shipboard duty?" In response, Robinette (1981) stated: "A search for men of similar educational attainment, aptitude for technical work, and training by the Navy in their occupation, led to the selection of the equipment operators as members of the potential low-exposure cohort. The duties of these men required, for the most part, that they be below decks in areas where equipment was not being repaired and which were not traversed by operating radars."

Lester and Moore (1982a) had postulated that prolonged, repeated exposure to weak RFEMF might be associated with increased cancer incidence. Because radars have been in operation at military air bases since World War II, the authors had hypothesized that detectable increases in cancer mortality might be found in areas surrounding Air Force bases. To test this hypothesis, they searched "Guide to Air Force Bases" (Air Force Magazine, 1969) to locate such bases (AFBs) in the continental United States that had been operational in the period 1950-1969, and found 92 counties that had at least one AFB. They then obtained the population in each of those counties from the 1960-census data (CENSUS, 1967), and selected a control county for each AFB county, defined as the one in the same state that was closest in population (larger or smaller) that did not have an AFB. For the AFB counties, the mean population was 237,684 ± 254,683 (SD); the values for the non-AFB counties were 209,893 ± 242,128. The difference between the two distributions was not statistically significant.

The authors indicated that data on civilian air bases for that period were not available. They also stated: "It should be noted that many counties in both groups had other air bases. Also, the counties varied considerably in geographic and economic characteristics. These factors would tend to bias the data against the hypothesis. Despite this confounding, the design demands that the presence of an AFB produce sufficient electromagnetic effect that it would be relatable to a higher cancer mortality in that county. No attempt was made to assess the possible role of other carcinogens."

Cancer mortality ratings (deaths from all types of cancer) for the AFB and control counties were obtained from the "Atlas of Cancer Mortality for U.S. Counties: 1950-1969" (HEW, 1975). The authors adjusted the mortality data for age, and numerically indexed them in the following five categories relative to cancer mortality in the general U.S. population:

MORTALITY RANKING	LESTER AND MOORE INDEX
Significantly high, in highest decile	4
Significantly high, not in highest decile	3
In highest decile, not significant	2
Not significantly different from U.S.	1
Significantly lower than U.S.	0

Data were available separately for males (M) and females (F). Lester and Moore (1982a) presented the results shown in Table 12 (their Table 1):

TABLE 12: CATEGORIES OF CANCER MORTALITY
[Lester and Moore (1982a)]

MALES AFB Counties	MALES nonAFB Counties	FEMALES AFB Counties	FEMALES nonAFB Counties	INDEX
21	12	13	7	4
2	1	4	2	3
5	6	0	3	2
16	21	33	34	1
48	52	42	46	0
92	92	92	92	

From those results, the authors made the following assumptions:

a. Categories 4 and 3 can be combined as significant incidence (+); categories 1 and 0 can be combined as nonsignificant incidence (-).
b. Category 2 can be deleted.
c. Since there was an effort to match counties by population, the proper statistical analysis is a test for correlated proportions comparing AFB with nonAFB counties.

The authors then reclassified their data in pairs as shown in Table 13 (their Table 2):

TABLE 13: RECLASSIFIED CANCER MORTALITY DATA
[Lester and Moore (1982a)]

INCIDENCE	MALES	FEMALES
AFB (+) and nonAFB (+)	9	7
AFB (-) and nonAFB (-)	57	70
AFB (+) and nonAFB (-)	12	10
AFB (-) and nonAFB (+)	4	2
TOTALS	82	89

Their "test for correlated proportions, corrected for continuity, one-tailed test" of the data in Table 13 yielded $p = 0.04$ for males and $p = 0.02$ for females, from which they concluded that AFB counties, had a significantly higher incidence of cancer mortality for the period 1950-1969 compared with population-matched nonAFB counties. As presented, however, their results do not confirm that increased cancer mortality is associated with RFEMF exposure, but only that such mortality appears to be correlated with the presence of an operational AFB.

The test for a statistically significant difference between the AFB counties and control counties depends heavily on how well matched were the control counties with the AFB counties in all factors except the presence of an AFB. The AFB identities were not given in the paper, so on inquiry, Dr. J. Lester kindly made available the raw data. A review of those data revealed several apparent

inconsistencies, so Polson and Merritt (1985) did an independent analysis, using the list of AFBs provided by Dr. Lester and the methodology described in the paper.

For the independent analysis, the Rand McNally "1982 Commercial Atlas and Marketing Guide" (Rand McNally, 1982) was used to ascertain the counties in which the specific AFBs were located. The populations of those counties were determined from CENSUS (1967), the reference used by Lester and Moore (1982a), and the control counties closest in population in the same state were identified. The cancer-mortality categories for males and females in the control and AFB counties were obtained from HEW (1975), the source also used by Lester and Moore. On reassembly of the raw data, the following emerged:

1. The total number of AFB counties was reduced to 91 because one county was counted twice (Luke AFB and Williams AFB are in the same county).

2. Of those 91, Lester and Moore located 13 AFBs in incorrect counties.

3. In 43 of the remaining 78 cases, Lester and Moore either used the incorrect control county (as defined above) or incorrectly assigned the M or F category to the control county.

4. Of the remaining 35 cases where Lester and Moore had selected the AFB and control counties correctly, there were 22 cases (16 counties) for which they had incorrectly assigned the M or F categories.

Thus, of the original 92 AFB/control county pairs of data used by Lester and Moore, only 19 and their M and F categories appeared to be correct. Use of the corrected data for the 91 AFB counties yielded Table 14:

TABLE 14: REVISION OF TABLE 13
[Polson and Merritt (1985)]

MALES AFB COUNTIES	MALES non-AFB COUNTIES	FEMALES AFB COUNTIES	FEMALES non-AFB COUNTIES	INDEX
16	10	9	7	4
0	1	3	3	3
6	8	0	2	2
17	24	33	36	1
52	48	46	43	0
91	91	91	91	

Reclassification of these data based on the assumptions of Lester and Moore yielded Table 15:

TABLE 15: REVISION OF TABLE 14
[Polson and Merritt (1985)]

INCIDENCE	MALES	FEMALES
AFB (+) and nonAFB (+)	6	6
AFB (-) and nonAFB (-)	58	73
AFB (+) and nonAFB (-)	10	6
AFB (-) and nonAFB (+)	6	5
TOTALS	80	90

A statistician was consulted about the "test for correlated proportions" used by Lester and Moore. A test by this name does not appear in any of the references familiar to the statistician. However, McNemar's Test (Fleiss, 1981) is suitable and is similar to the test by Lester and Moore because use of this test yielded the same z and p values cited by Lester and Moore for Table 14 (their Table 2).

McNemar's Test of the data in Table 15 yielded $z = 0.7$, $p = 0.23$ for males and $z = 0$, $p = 0.50$ for females, both nonsignificant.

In summary, an independent reanalysis by Polson and Merritt (1985) of the data for the 91 AFB counties found by Lester and Moore (1982a) and their population-matched control counties not having an AFB did not confirm their finding. Instead, for the period 1950-1969, the AFB counties had incidences of cancer mortality for males or females that did not significantly differ statistically from those in counties without an AFB most closely matched in population. The significance original finding by Lester and Moore (1982a) apparently was the result of an incorrectly assembled database. The reevaluation by Polson and Merritt (1985) was published in the same journal as the Lester and Moore (1982a) paper, together with a response by Lester (1985), who took issue with some aspects of the reanalysis and did not accept the revised findings

In another study, Lester and Moore (1982b) sought to determine whether there was a geographic pattern of cancer incidence within the city of Wichita, Kansas, and whether specific sources of RFEMF could be identified and related to any such pattern. The authors reported that they had found a neighborhood pattern of cancer incidence in that city, with the suggestion of a time element in its appearance, and noted that cancer tended to occur on leading terrain crests relative to radar transmissions, and was less frequent in the valleys. They presented a formula that related incidence of cancer to the terrain and the presence of RFEMF, from which they derived an overall finding that cancer incidence in Wichita appears to be related to the probability of exposure to radar.

At that time, Wichita had a population of 262,766 (from a 1973 census). With the exception of two industrial areas, the city was divided into 94 census tracts of approximately equal population. However, Lester and Moore (1982b) included only 76 of the tracts in their analysis. The Department of Community Health had stratified the tracts into three health and economic classes (good, fair, poor), based on determinants such as levels of education, income, crowding, unskilled workers, infant mortality, venereal disease, tuberculosis, and housing. Regarding freedom from air pollution, Wichita then ranked second out of 52 major cities of over 80,000 population in the United States, thus presumably removing that agent as a possible confounding factor.

Wichita lies on essentially a flat plain bisected by the Arkansas River, with two low ridges (100-ft and 20-ft changes in elevation) to the west and northwest of the city limits. Wichita Mid-Continent Airport lies 9.7 km southwest and 35 ft higher than the city center. McConnell AFB is located 7.2 km southeast and 130 feet higher than the city center.

The authors obtained morbidity data on all first diagnosed cancer cases of Wichita residents for 1975, 1976, and 1977 from five city hospitals, for a total of 3004 cases for the three years. The ratio of the number of diagnosed cases to the tract population for each year yielded the incidence rates for the 76 census tracts considered. They then analyzed the data on the incidence rates, age, economic stratification (of the residence tract of each person), male/female ratio, and race, and obtained a correlation matrix for those 76 tracts. They also obtained mortality data for all cancer deaths of Wichita residents for those three years, and similarly analyzed those incidence rates to obtain another correlation matrix for the 76 census tracts.

The formula derived by the authors for treating the data was based on the following hypotheses:

(1) The major contributors to RFEMF exposure were the radar transmitters at the airports.

(2) Radar exposure is by line-of-sight.

(3) RFEMF exposure correlates directly with elevation.

(4) Any shielding by intervening terrain confounds the elevation exposure criterion.

This paper is replete with flaws, the most serious of which is that the authors, "to find a possible connection between cancer incidence and external electromagnetic fields", assumed that the population is exposed only to the RFEMF from radars at the two airports adjacent to the city. They provided no measurements to support this assumption and gave no indication that the scan sectors of such radars were considered. It is noteworthy, discussed in Section 2.1 above, that the Environmental Protection Agency (EPA) had measured the ambient RFEMF levels in 15 major cities around the United States, which showed that by far the major contributors to environmental levels of RFEMF are FM and TV broadcast transmitters, not radar systems. Although the EPA did not include Wichita among the cities it surveyed, it would be reasonable to assume that the RFEMF environmental situation there differed little from those of the 15 cities surveyed.

Even if the assumption above that the radars were the primary sources of exposure were correct, a model based on the physical laws of RFEMF propagation should have been used, specifically the inverse-square-law of attenuation with distance from the sources and the shielding effects of artificial structures and buildings and of the terrain. Other flaws in the paper were arbitrary assumptions for the heights of the radars and the false precision imparted by citing the city and airport elevations to five significant figures ("average" elevations accurate to within 5 mm).

Unfortunately, the propagation and exposure model used by the authors bore no relationship to such factors, and any conclusions drawn therefrom are unrelated to the actual exposure levels. Instead, the results of this paper appear to be a good example of spurious correlation. Any relationship between radar exposure and cancer incidence, if such existed, was not demonstrated by the data and analysis presented in this paper.

In a correspondence item, Milham (1982) presented some data that showed higher than expected mortality rates from "all leukemia" and from "acute leukemia" for men in occupations that presumably involved exposure to electric and magnetic fields. The author ascertained expected numbers of deaths from 158 causes (including various forms of leukemia) in white males 20 years of age and older who resided in Washington State during 1970 through 1979, and standardized them by age and year of death. He then determined the number of deaths from each cause in each of 218 coded occupations and calculated the ratio of the number of deaths from each cause in each occupation to the expected number of deaths from that cause, the "proportionate mortality ratio [or rate]" (PMR). He analyzed a total of 438,000 deaths in that manner.

Statistical treatment of the data was not given, but the PMRs for seven of the 11 occupations assumed to have involved exposure to electric and/or magnetic fields were marked as not statistically significant. However, for the four other occupations (electricians, television and radio repairmen, power-station operators, and aluminum workers), the PMRs for all-leukemia were 138%, 157%, 259%, and 189%, respectively, of which three occupations were marked significant at the $p<0.01$ level. The exception was for the television and radio repairmen, for which there were only 5 cases versus 3.2 expected, numbers too small to render their ratio statistically significant. The PMRs for acute leukemia in the same four occupations were 178%, 291%, 282%, and 258%. Again, three of the PMRs were marked significant ($p<0.01$), but the nonsignificant exception was for power-station operators. The author surmised that the significant increases in leukemia mortality were associated primarily with exposures to large dc electric currents and high alternating electric and magnetic power fields.

Liburdy (1982) took issue with the Milham (1982) finding, citing several references that do not support it, including a NIOSH review on carcinogenesis and nonionizing radiation by Dwyer and Leeper (1978). Liburdy (1982) also noted: "The report cites over 60 quantitative studies that identify mutagenic, teratogenic, developmental, hematologic, and neurologic effects. Although these phenomena are implicated in the process of carcinogenesis, cancer was not observed...Negative results do not conclusively rule out a health risk; the data, however, argue against an association."

The correspondence item above by Milham (1982) described one aspect of a much larger study by Milham (1983), in which he analyzed information on age and year of death in Washington State of 429,926 male decedents for 1950-1979 and 25,066 female decedents for 1974-1979. Presented were

cause-of-death analyses (160 causes) for 219 male and 51 female occupational categories. The author, referring to the Registrar General of England and Wales, stated:

"The Washington State mortality pattern is, in general, consistent with both the Registrar General's results and with the published literature. Some of the new occupational mortality findings published in the 1950-1971 report and in this updated version have been confirmed. Others warrant follow-up. These include a lung cancer excess in workers at the ASARCO Tacoma copper smelter, increased mortality due to multiple myeloma and pancreatic cancer in workers at the Hanford atomic energy facility, and excess mortality due to cancer of the pancreas, lymphoma, leukemia, and emphysema in aluminum workers.

"New findings in this report are a leukemia increase in workers exposed to electric and magnetic fields and a deficit of multiple sclerosis deaths among outdoor workers."

In this report, 20 microfiche negatives were provided in addition to 167 pages of text; these microfiches contained the detailed raw mortality data and the calculated proportional mortality ratios (PMRs) for males and females by gender occupations. (The occupation of each decedent was obtained from the statement on his/her Washington-State death certificate.) Also contained in the microfiches was additional information on occupations ranked by PMR within each cause of death, and causes of death with significantly ($p<0.05$) elevated or reduced PMRs by occupation, both for the males only. Eighty pages of the printed report described the occupation codes (common for both men and women) used in filling out the death certificates, the occupation groupings for men, the occupation groupings for women, an index of occupations for men, and an index of occupations for women.

Milham's (1983) analysis progressed from the raw mortality data through successive clusterings of the data in like occupational groupings, presumably to obtain sufficient numbers in each cluster to permit meaningful statistical analysis. As in Milham (1982), the proportionate mortality ratio (PMR) was used. Epidemiologists Lilienfeld and Lilienfeld (1980) have stated (pp. 74-75): "The proportionate mortality rate does not directly measure the risk or probability of a person in a population dying from a specific disease as does a cause-specific mortality rate." Moreover, the more common method is the "standardized mortality ratio" (SMR), used by the Registrar General as described on pages 78-80 in Lilienfeld and Lilienfeld (1980). It represents the percentage of actual deaths for each cause relative to the expected number of deaths from that cause, independent of any other SMR.

Milham's printed report contained 63 pages of one-paragraph commentaries that described the mortality pattern in each of the occupational groupings as seen by the author. There were 219 such commentaries for males and 51 for females. Some of the commentaries appear to be highly subjective and to reflect the personal biases of the author. For example, the commentary below on female mortality in one occupation was given on page 63:

"Waitresses
Occupation code 875
Total deaths 862

"Cancers of the esophagus, stomach, larynx, lung, cervix and uterus unspecified have increased PMRs. Psychoses, pulmonary emphysema, cirrhosis of the liver, motor vehicle accidents, and homicide have mortality increases. Much of this mortality pattern may be due to life-style patterns, i.e., smoking, drinking, and promiscuity."

Other commentaries seem to include some elevated but nonsignificant PMRs because the author apparently believed that they contribute to the overall mortality pattern considered appropriate by him for that occupation. For example, page 54 has the following commentary:

"Dietitians and Nutritionists
Occupation code 073
Total deaths 104

"These women show a significant excess of malignant neoplasms of the digestive organs (PMR=254 based on 7 deaths) in the 20-64 age class. This is due to 4 deaths observed from cancer of the pancreas to less than 2 expected. Malignant neoplasms of lymphatic and hematopoietic tissues (age 20-64) show a PMR of 476 based on 5 deaths, 3 of which were in the other lymphoma category. Diabetes mellitus had a PMR of 225 based on 5 deaths."

It is seen that of 104 total deaths in this occupation for women, the author had selected 17 deaths and presented them as though dietitians and nutritionists might be expected to die more often of malignant neoplasms of the digestive organs and diabetes mellitus. The other 87 deaths were ignored. This clearly demonstrates a subjective and selective bias in the author's application of the PMR analytical technique.

The author carried the analysis one step further by examining mortality by cause of death and by showing occupational groupings with statistically significant elevations or reductions in PMR. There are several problems with some of the patterns that emerged in this treatment. On page 67, for example, in the commentary on male mortality by occupation within cause-of-death groups is the following:

"Malignant Melanoma of Skin (ICD 190)

"Three of the four occupations with high PMRs (clergymen, school teachers, hotel managers) have no obvious relationship to outdoor work or exposure to sunlight. Navy and Coast Guard personnel, however, probably are exposed to sunlight."

This presumption that Navy and Coast Guard personnel have elevated PMRs for malignant melanoma of the skin because they are exposed to sunlight would seem to indicate a considerable bias on the part of the author. He did not indicate why these personnel would spend more time in the sun than school teachers, for instance, other than his personal belief that they do so.

In the next step in the analysis, Milham examined the mortality patterns of groups of selected occupations that appeared to have similar environmental exposures. It is here, on one page (75) of the entire report, that categories of workers presumed to be occupationally exposed to magnetic and/or electrical fields are juxtaposed and PMRs for two categories of leukemia (acute leukemia and all leukemia) are presented. Shown were the following 11 occupations: electrical engineers, electronic technicians, radio and telegraph operators, electricians, power and telephone linesmen, television and radio repairmen, motion picture projectionists, aluminum workers, streetcar and subway motormen, power station operators, and welders and flame cutters.

Of the 22 categories for the two leukemia categories by 11 occupations, there were 3 cases where the PMR was significant at the 1% level, plus 2 cases at the 5% level. The other PMRs, though elevated in 13 cases and depressed in 3 (with 1 the same), were not statistically significant. The significant cases, electricians (both categories of leukemia), aluminum workers (both categories of leukemia), and power station operators (all-leukemia only), comprised 79 of the total of 136 leukemia deaths actually observed in those occupations. The excess number of deaths (observed minus expected) for those three occupations was 28. On page 5, the author stated the following on the PMR technique:

"The major flaw of the proportionate mortality ratio (PMR) is that it says nothing about total force of mortality for a given occupation... All occupations have a total PMR of 100. Also, since the cause-of-death specific PMRs must sum to 100, a very high or low PMR in a common cause-of-death group will affect the other PMRs for that occupation."

In other words, by virtue of the technique itself, the 5 significantly high PMRs mentioned above might have arisen because other PMRs in three of the 11 occupations were abnormally low. In view of this point, little credence can be given to the author's claim that the increased PMRs for all leukemia and for

acute leukemia are associated with exposure to electric and magnetic fields. The other point to be made is that there must be a dose-response relationship to conclude that cause-and-effect applies in this or in other epidemiologic studies. At best, such a relationship is unproven here; without exposure data for the individuals or even for the occupations, it is solely an assumption that persons in these occupations actually do experience greater exposure to electrical and magnetic fields than do those in other occupations. For example, electricians, the occupation with the largest number of leukemia deaths (51), actually spend a large part of their time working on circuits that are not energized.

Perhaps the strongest overall criticism of this epidemiologic study is that it illustrates an approach that statisticians commonly refer to as "data mining", in which a very large data base is "picked over" for "nuggets" of (locally) statistically significant items, which are then assembled to show purported relationships. The usual statistical methodology is reversed: Statistical significance is found first and then hypotheses are formulated. However, epidemiologists do recognize and characterize such studies as appropriate for "hypothesis generation", but give little credence to any specific quantitative findings therefrom; instead, they give more credence to specific studies in which hypotheses, however derived, are tested a priori.

In summary, the claim in Milham (1983) that workers who had been exposed occupationally to electric and magnetic fields showed increased incidence of all leukemia and acute leukemia is weak at best and questionable in the context of the full report on occupational mortality in Washington State by the author. The methodological approach used did not meet the usual criteria for statistical testing of hypotheses: no hypotheses were assumed a priori. The commentaries on the patterns of mortality underlying the different occupations from which the groups were selected, such as workers with electric and magnetic field, seemed to show personal bias by the author. Also, the PMR technique could have yielded an apparent increase in the PMR in one cause-of-death category from an abnormally low PMR in another category. (These criticisms also apply to the cause-and-effect relationships claimed in the report for other agents and occupations considered.)

Wright et al. (1982), in a correspondence item, presented tabulated data on "proportional incidence ratios" (PIRs) for male cases of leukemia in the Cancer Surveillance Program of Los Angeles County for the years 1972 to 1979. The results were partitioned into "all leukemia", "acute leukemia" and "acute myelogenous leukemia" (AML) for 11 occupations presumed to involve exposure to electric and magnetic fields. They reported a trend toward increased PIRs in those occupations, with the highest risk for AML, but stated: "The occupations grouped as sharing exposure to electric and magnetic fields undoubtedly share other exposures. While significant exposure to ionising radiation is probably not present in most of these jobs, metal fumes, solvents (including benzene), fluxes, chlorinated biphenyls, synthetic waxes, epoxy resins, and chlorinated naphthalenes are other exposures that may be shared." Thus, their findings provide little if any credence to an association of leukemia with exposure to electromagnetic fields. They did include "television and radio repairmen" as one of the occupations examined but had found only one case of leukemia in that occupation, a point relevant to RFEMF (as defined herein).

McDowall (1983) (also in a correspondence item) noted the findings of Milham (1982) and Wright et al. (1983), and presented the PMRs from his independent analysis of leukemia mortality for males 15-72 years of age from 10 electrical occupations during 1970 to 1972 in England and Wales. Shown in Table I of the paper were the PMRs and numbers of cases of all leukemia, lymphoid leukemia (all and acute), and myeloid leukemia (all and acute) in 10 categories of electrical occupation. The author indicated that taken collectively, the mortality distribution for all those occupational categories did not significantly differ from expected. The PMRs ranged from 61 for "radio and radar mechanics" to 249 for "telegraph radio operators". The PMRs for acute myeloid leukemia (AML) did exceed 100 for four occupations: 213 for "professional electrical engineers", 231 for "self-described electrical engineers", 241 for "telegraph radio operators", and 305 for "professional electronic engineers". The author did note that PMRs are often suspect, but may be of value, especially if very different from 100.

This author also did a case-control mortality study of 537 males of ages 15 years and up from AML in the year 1973, with 1,074 randomly selected male decedents in the same age range from all causes except leukemia as controls. The results were displayed in Table II of the paper. In the category "all

electrical occupations" (30 of 36 AML cases), the relative risk (RR) was 2.1, with (95%) CI=1.3-3.6, which was significant. The RRs for the five specific subcategories of "all electrical occupations" did exceed 1.0, but all of their respective CIs spanned 1.0, rendering the RRs nonsignificant. These results are at variance with the author's comment of increased risks for those in electrical occupations. Thus, little if any credence can be given to the findings in this brief paper.

Coleman et al. (1983), noting the studies by Milham (1982), Wright et al. (1982), and McDowall (1983), reported on a study of leukemia incidence among men in 10 electrical occupations in South-East England. The authors indicated that the South Thames Cancer Registry listed about 30,000 tumors a year in a population of 6.5 million, and they calculated the "proportional registration ratio" (PRR) [percentage observed/expected] for leukemia in males of ages 15-74 years during 1961-1979. They compared the number of registered leukemias in the males of each 5-year age group in each of the 10 electrical occupations examined by McDowall (1983) with the number expected. Their assumption was that the leukemias in each electrical occupation is in the same proportion to other cancers as for the men with leukemia in all occupations (their null hypothesis).

Their analysis indicated a 17% excess of all leukemias in all electrical occupations (113 cases versus 96.5 cases expected: PRR=117, 1-sided $p<0.05$). However, even though 8 of the 10 occupations showed PRRs exceeding 100, the occupation "radio/radar mechanics" showed a deficit for all leukemias (1 in 22 cases versus 4.63 expected; PRR=22, $p=0.07$). The PRRs for all electrical occupations also showed no excess of chronic myeloid leukemia (PRR=96), but there were a 46% excess of acute lymphoid leukemia, a 29% excess of chronic lymphoid leukemia, and a 23% excess of acute myeloid leukemia (AML). The excesses of AML were in 7 of the 10 occupations, but not all of those PRRs were significant. Moreover, there were no cases of AML in the occupations "radio/radar mechanics", "professional electronic engineers", or "assemblers (electrical equipment)", and the numbers of cases in the other occupations were small. For example, the highest PRR, 195, was for "electrical/electronic fitters", but there were only 5 cases. Indeed, as indicated above, the total number of leukemia cases in all electrical occupations was only 113 out of 3200 leukemia cases for all occupations.

In a brief report, Milham (1985) described his study of mortality in male members of the American Radio Relay League (ARRL), a group of amateur radio operators presumably exposed to RFEMF while they were operating their transmitters. For the years 1971-1983, 296 male-member deaths in Washington State and 1642 in California were listed in QST, the monthly ARRL publication. Death certificates were obtained for 280 (95%) of the Washington decedents, and information on the age, date, and cause of death was obtained for 1411 (86%) of the California decedents, a total of 1691 deaths. A PMR of 281 was found for acute, chronic, and unspecified myelogenous leukemia (16 deaths found versus 5.7 deaths expected, $p<0.01$); the PMR for monocytotic leukemia was only 77 (well below 100), and was 0 for lymphatic leukemia. Thus, the PMR for all leukemias was 191 (24 deaths versus 12.6 deaths expected, $p<0.01$). The author noted that many of the ARRL members are employed in occupations involving exposure to electromagnetic fields, but that occupational exposure alone does not explain the excess deaths.

Wangler et al. (1985) disagreed with Milham's (1985) suggestion that there may be a greater than normal leukemia risk in amateur radio operators, based on several points. First, those authors questioned the selection of radio amateurs to test that hypothesis. They cited a 1980 survey in Canada and the U.S. that showed that typical amateurs spent 6.1 hours per week on that activity, much of which was highly variable and most of which involved listening, with intermittent transmissions at low average radiated powers. Second, they cited Milham's statement that 35% of the amateurs in Washington State had been employed in electrically related occupations, and queried how many of the higher leukemia deaths were occupationally related. Third, they noted that the "silent keys" are not representative of the amateur radio community as a whole because those decedents are listed in QST only when reported by a family member. Fourth, they noted the possibility of exposures of the decedents to toxic chemicals. Last, they questioned the statistical treatment used by Milham (1985).

Coleman (1985) took issue with the comments by Wangler et al. (1985) regarding Milham's (1985) statistical methodology and conclusions, except for the point about how representative of the amateur radio community were the numbers of decedent "silent keys".

For the reasons stated previously, use of the Proportionate Mortality Ratio (PMR) or similar statistical measures by Wright et al. (1982), McDowall (1983), Coleman et al. (1983), and Milham (1985) provides little if any basis for ascribing any credence to their findings.

In a subsequent study, Milham (1988a) examined the mortality data for a larger number of amateur radio operators. The author extracted the names of 67,829 males in Washington State and California listed as licensed in the 1984 U.S. Federal Communications Commission Amateur Radio Station and/or Operator File between 1 January 1979 and 16 June 1984. Those names were searched for deaths during the five-year period from 1 January 1979 to 31 December 1984, which yielded a total of 2,485 male decedents taken to have had 232,499 person-years at risk. Herein, the author used the SMR rather than the PMR. For an SMR exceeding 100, whether the excess number of deaths is significant would depend on the size of the 95% confidence interval (CI) for that SMR and if the CI does not include 100 within its range. Similar considerations apply for a smaller than expected number of deaths for an SMR less than 100.

The total number of expected deaths in both states from all causes was 3,479, so the 2,485 deaths of licensees yielded an SMR of 71, with a CI of 69-74, indicating death rates for licensees that were significantly lower than in the general population. The highest number of deaths found was 1,208, in the category "all circulatory diseases". However, 1,732 deaths were expected, so the SMR was only 70 (CI = 66-74), also indicating a significantly lower death rate than in the general population.

In the category "all malignant neoplasms," there were 741 deaths versus 839 expected, yielding an SMR of 89 (CI = 82-95), again a significantly lower death rate than for the general population. The only subcategory of malignant neoplasms that yielded an SMR that significantly exceeded 100 was for "other lymphatic tissue", in which there were 43 deaths versus 27 expected. The SMR was 162, with a CI of 117-218.

The subcategory "leukemia" had 36 deaths versus 29 expected, for an SMR of 124, but the CI, 87-172, spanned 100, thus indicating that this finding was nonsignificant. The author also considered 9 subdivisions or sub-subdivisions of "leukemia" and found that of the 36 deaths, 15 were for "acute myeloid leukemia" versus 8.5 of the expected 29. These values yielded an SMR of 176 with a CI of 103-285, a statistically significant result. However, little if any credence can be given to this finding, in view of the small numbers of deaths relative to the actual and expected totals. Thus, despite any claims to the contrary by the author, the results of this study do not offer any confirmation of those in Milham (1983).

In a later brief paper, Milham (1988b) also presented an analysis of the mortality data for amateur radio operators in Washington State and California by the five license classes of the Federal-Communications-Commission: novice, technician, general, advanced, and extra. The novice class covered the usual entry-level or beginners license, and the other four classes included the operators with successively increasing technical knowledge and skill (Morse-code speed). The mean ages of the licensees in each class were 38.4, 44.3, 49.5, 51.4, and 49.2 years, respectively, with an overall mean of 46.8 years. Table 16 (adapted from Table 1 of the paper) shows the numbers of deaths and SMRs in each class from all causes, all malignant neoplasms, and various specific types of malignancies. The author did not provide the 95% confidence intervals, but did tag the SMRs considered significant ($p<0.05$).

TABLE 16: MORTALITY IN AMATEUR RADIO OPERATORS BY FCC LICENSE CLASS
[Milham (1988b)]

Cause of Death	Novice (n=12,279)		Technician (n=13,702)		General (n=18,649)		Advanced (n=17,436)		Extra (n=5,763)		All (n=67,829)	
	Deaths	SMR	Deaths	SMR	Deaths	SMR	Deaths	SMR	Deaths	SMR	Deaths	SMR
All Causes	247	61*	409	80*	862	81*	798	69*	169	49*	2,485	71*
All Malignant Neoplasms	78	81	117	94	236	92	253	90	57	73	741	89
Brain Cancer	1	34	4	112	11	175	11	174	2	114	29	139
Lymphatic and Hematopoietic Neoplasms	9	101	18	163*	26	119	27	115	9	134	89	123
All Leukemias	5	139	7	160	10	114	10	105	4	145	36	124
Myeloid Leukemia	1	43	6	230	5	141	5	151	1	91	18	140
Multiple Myeloma and Other Lymphomas	3	96	8	199	15	184*	13	147	4	161	43	162*

*p<0.05

Table 16 indicates that the SMRs for all causes of death were all significantly less than 100. Also, for deaths from all malignant neoplasms, the SMRs for all five license classes were below 100 and nonsignificant, but the collective SMR for that category was 89 and marked significant. As in Milham (1988a), both results may be an indication that amateur radio operators are healthier than the general population.

On the other hand, the SMRs for lymphatic and hematopoietic neoplasms all exceeded 100, but the only significant excess was for the technician-license class, a result that rendered significant the collective SMR for that death category. Similarly, except for the novice class, the SMRs for multiple myeloma and other lymphomas exceeded 100, but the excess was significant only for the general-license class, again rendering the collective SMR for that death category significant. In both cases, however, the numbers of deaths were small: 18 of 409 deaths in the technician-license class and 15 of 862 deaths in the general-license class. Thus, as for the previous studies by this author, the findings above and the author's interpretation thereof are open to question.

Pearce et al. (1985), citing their then unpublished case-control study of possible relationships between leukemia incidence and various agricultural occupations in New Zealand, reported leukemia excesses in specific occupations "with potential for exposure to electrical and magnetic fields associated with alternating current". However, the authors also included occupations that may have involved exposure to RFEMF frequencies, as indicated below. That study was of 546 male leukemia patients registered during 1979-1983 at ages 20 years and older, and the controls were 2,184 men chosen from the New Zealand Cancer Registry, with 4 controls per case matched on age and registration year.

In this brief paper, the authors extracted and tabulated the leukemia odds ratios (ORs) and 95% confidence limits (CIs) for those in the following specific occupations: electrical and electronic engineers, electrical and electronic technicians, electrical fitters, electronic equipment assemblers, radio/television repair, telephone installers, linemen, and power-station operators. There were 18 of the 546 leukemia cases in those occupations. The controls totaled 43 persons. Statistically significant leukemia excesses were shown for the electronic equipment assemblers (4 cases versus 0.5 expected, for an OR of 8.17, a CI of 1.49-44.74, two-tailed p=0.02), and in the radio/television repair category (7 cases versus 1.5 expected, OR=4.75, CI: 1.59-14.23, two-tailed p=0.01). [As noted below, the occupations for these two results were stated erroneously in the table.] There was also a nonsignificant excess in the power-station operators (1 case versus 0.3 expected, OR=3.93, CI: 0.25-63.08, p=0.33). The authors suggested two hypotheses for the excesses: (1) exposure to nonionizing radiation, and (2) exposure to metal fumes and other substances used in electrical components or their assembly.

In a brief correction paper, Pearce (1988) noted that the excesses above should have been ascribed to the occupations "radio/television repair" and "electricians", respectively, and that there were no cases in the "electronic equipment assemblers" category. The corrected results are shown in Table 17 (adapted from both papers). The author noted that some of the additions are approximate because of rounding.

TABLE 17: ODDS RATIOS FOR LEUKEMIA IN SPECIFIC OCCUPATIONS
[Pearce (1988)]

Type of Electrical Work	Exposed Cases	Expected Cases	Controls	Odds Ratio (95% CI)	2-Tailed P-Value
Electrical/Electronic Engineers	1	1.3	5	0.79 (0.09-6.79)	0.83
Electrical/Electronic Technicians	1	1.0	4	1.04 (0.12-9.37)	0.97
Electrical Fitters	2	3.0	12	0.67 (0.15-3.02)	0.61
Electronic equipment Assemblers	0	---	0	-------	----
Radio/television Repair	4	0.5	2	8.17 (1.49-44.74)	0.02
Electricians	7	1.5	6	4.75 (1.59-14.23)	0.01
Telephone Installers	0	0.3	1	--------	0.74
Linemen	2	3.0	12	0.66 (0.15-2.96)	0.59
Power-Station Operators	1	0.3	1	3.93 (0.25-63.08)	0.33
TOTALS	18	10.8	43	1.70 (0.97-2.97)	0.06

As seen above, significant excesses were reported for radio/television repair and electricians, which, along with citations to the findings of Milham and others, led the authors to give greater credence to hypothesis (1) above: exposure to nonionizing radiation. However, the findings are questionable from a statistical viewpoint, primarily because of the small numbers of cases. Moreover, without any valid rationale, the authors ignored the nonsignificant excesses (and decreases) in leukemia cases in the other occupations they designated as "electrical" (and therefore possibly subjected to exposure to electric and magnetic fields), which yielded a nonsignificant overall OR. Another point was their scientifically unjustified display of the ORs and CIs to three or four significant figures, thus conveying an impression of higher accuracy for the findings than is warranted.

Pearce et al. (1989) described a more extensive case-control study of electrical workers in New Zealand. The study involved 19,904 male patients with all types of cancer registered during 1980-1984 at ages 20 years and older at time of registration and whose occupations were recorded (about 80% of the total number of cases). The analysis was focused on those with the electrical occupations considered in the previous study, and some patients previously considered were included in the present study. The control group for those with cancer at any specific body site (or cancer type) consisted of those with cancer at other sites.

There were 488 cancer cases in those electrical occupations. Of the 22 sites or cancer types tabulated, half had ORs less than 1.0 and the other 11 had ORs in the range 1.0 to 1.62. However, all of those 11 sites had CIs that spanned 1.0 (thus were nonsignificant), except for leukemia, which had a total of 21 cases versus 13 expected, OR=1.62 and a CI of 1.04-2.52 (significant). Within the specific occupations, the largest OR (7.86, CI: 2.20-28.09) was for radio/television repair, apparently confirming the previous finding. However, contrary to the previous finding for electricians, the OR (1.68, CI: 0.75-3.79) was nonsignificant. Also noteworthy was no excess of brain cancer (12 of 488 cases, OR=1.01, CI: 0.56-1.82).

The authors also examined the leukemia incidences within five subtypes: acute lymphatic, chronic lymphatic, acute myeloid, chronic myeloid, and "other" for the age group 20-64 years (9 cases total) and for the age group 65 years or older (12 cases total). The only significant excess was for chronic lymphatic leukemia (4 cases in the younger age group) rather than for acute myeloid leukemia, found in other studies.

The findings of this study are questionable for the same reasons as for the previous studies by these authors. In this paper, however, they did note that many of the comparisons involved small numbers of cases.

Brownson and Reif (1988) performed a study also based on the New Zealand Cancer Registry. However, the focus primarily was on agricultural workers, without any apparent potential relationship to RFEMF exposure.

Reif et al. (1989), in yet another study based on the New Zealand Cancer Registry, analyzed the incidences of brain cancer by six major occupational categories (Table 2 of the paper). The incidences for two of those categories were significant: for "agriculture/forestry/fishing" (79 of 452 cases), the OR was 1.38 with a CI of 1.08-1.77, and for "professional/technical" (74 of 452 cases), the OR was 1.32 with a CI of 1.02-1.69. The category "production workers", with a nonsignificant overall OR, was divided into 17 subcategories (Table 5 of the paper), one of which was "electrical worker", with a total of 8 of 185 cases. It had an OR of 0.78 with a CI of 0.39-1.59 (nonsignificant).

The "professional/technical" category was divided into 11 subcategories (Table 3 of the paper), including several that conceivably might have had some exposure to RFEMF (e.g., physical scientists and engineers). However, only two other subcategories clearly unrelated to RFEMF exposure showed significant brain-cancer excesses: "religious workers" (9 of 74 cases, OR=4.27, CI: 2.26-8.09) and "statisticians, mathematicians, and systems analysts" (4 of 74 cases) OR=4.26, CI: 1.51-11.98). Thus, this study based on the New Zealand Cancer Registry yielded no evidence of a relationship between the incidence of brain cancer and occupations that might have exposed the subjects to RFEMF.

Thomas et al. (1987) analyzed the risk of brain-tumor mortality for men occupationally exposed to MW/RF radiation (RFEMF), lead, and soldering fumes in the petrochemical industry. The authors obtained death certificates of men who had died at age 30 years or older from brain tumors or other tumors of the central nervous system between 1 January 1979 and 31 December 1981 in northern New Jersey and in Philadelphia and its surrounding counties. They acquired similar data for men who had died between 1 January 1978 and 30 June 1980 in Louisiana's gulf coast. The lifetime work histories for the case men were obtained from next-of-kin. One control for each case was selected from men matched in age and year of death and area of residence who had died from causes other than brain tumor.

The case men were classified regarding exposure to RFEMF by two methods. In the first method, the men were divided into two job-related categories: those involved in the design, manufacture, installation, or maintenance of electronic or electrical equipment, and those exposed to RFEMF in other types of jobs (such as welding and radio broadcasting). In the second method, a certified industrial hygienist assigned codes to each job in the occupational histories of the individuals for presumed exposure to: RFEMF, lead (high, moderate, or low), and soldering fumes (high or low). The authors noted that the RFEMF exposure classifications in the two methods overlapped considerably, but that the second method included men in supervisory jobs not considered as having been exposed in the first method.

Information was available on 435 cases and 386 controls. Of the cases, 300 men had *glioblastoma multiforme*, *astrocytoma*, and mixed gliomas with astrocytic cells, all of which were grouped into the category "astrocytic tumors"; 90 had other recognized types of tumor cell; and 45 had unrecognized types of tumor cell. The authors estimated the "maximum-likelihood relative risk" (RR) and the 95% confidence interval (CI) for each type of exposure and job category, and they adjusted the data for possible confounding influences of educational level (highest number of years of school completed). As in other studies, they regarded any RR greater than 1.0 as a significant excess if its CI did not span 1.0.

The analyses showed significantly elevated RRs for all brain tumors among the men classified as exposed to RFEMF (RR=1.6, CI=1.0-2.4); in jobs involving design, manufacture, installation, or maintenance of electronic or electrical equipment, the RR was 2.3 and the CI was 1.3-4.2. The RRs were not elevated for exposure to RFEMF in other types of jobs. The highest RR was for the combined classifications of engineers, teachers, technicians, repairers, and assemblers: RR=3.9, CI=1.6-9.9, with RR=4.6, CI=1.9-12.2 specifically for astrocytic tumor. RR increased with exposure duration by tenfold for

those in jobs associated with the manufacture and repair of electronic equipment for 20 or more years. For "tradesmen" (the combined categories of electricians, and power and telephone linemen), however, the RR was a nonsignificant 1.3 and showed no consistent pattern with increasing employment duration. On the other hand, the RRs were also higher for electronics workers classified as not having been exposed to RFEMF.

The RR for astrocytic tumors from exposure to soldering fumes was 3.4 with a CI of 1.6-7.5, but the variations with presumed exposure level were not large. However, nearly all of the men exposed to soldering fumes had such exposure in electronics manufacture and repair jobs. RRs were not elevated for exposure to lead by level (low, medium, high) or overall.

Based on those results, the authors suggested that simple exposure to MW/RF radiation was not the responsible agent for excess brain tumor risk. They noted that exposure to such radiation in electronics jobs is probably intermittent and may be accompanied by exposures to lead, solder fluxes, solvents, and other chemicals. They also stated that the results should be interpreted with some caution, because when they calculated the risks for specific occupations and for individual strata by employment duration, they obtained very small numbers in single cells of the tables.

The conclusions by the authors of this study appear to be correct, but the assumptions regarding the relationships between the various occupations and potential exposure to RFEMF are obscure, and some of the data apparently involved counting of subjects in more than one category. Thus, the positive and negative findings of the study are questionable.

Garland et al. (1987) cited reports that Hodgkin's disease may result from previous illnesses such as viral or non-viral infections, autoimmune disorders, preventive immunizations, or occupational exposure to various chemical agents. They remarked that an excess risk of Hodgkin's disease from exposure to [ionizing, primarily nuclear] radiation has not been established. They also noted that naval personnel perform a variety of occupational specialties and are subject to various environmental factors, such as crowding, that may foster the spread of infectious diseases that may increase the risk of Hodgkin's disease relative to the general population.

Those authors compared the rates of first hospitalizations for Hodgkin's disease in active-duty naval personnel during the years 1974-1979 with the incident rates of the general population derived from the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) database for the years 1973-1977 and with the total Navy rates. The overall results were an age-adjusted incidence rate of 2.9 cases per 100,000 person-years for the naval personnel and 3.7 cases per 100,000 person-years for the general population. By use of the standardized incidence rate (SIR) and 95% confidence limit based on the Poisson distribution, the difference was found to be nonsignificant. Although not noted by the authors, the occupations tabulated that may have involved exposure to RFEMF included "radioman", "electronics technician", "airman", "fire control technician", and perhaps others, showed no significant excess. The only statistically significant excess was for the occupational group "machinist's mate", a finding ascribed to exposure to various chemical and physical agents in close quarters.

Garland et al. (1988a) performed a similar study on the incidence of non-Hodgkin's lymphomas in active-duty, white-male naval personnel for the years 1974-1983, and found no excesses in any of the occupations tabulated. In actuality, the incidence of non-Hodgkin's lymphomas for the naval personnel was significantly lower than for the general population.

Garland et al. (1990) also studied the incidence of leukemia in white-male, active-duty naval personnel during the years 1974-1983, The data they examined were derived from the computerized Inpatient Follow-up Data System maintained at the Naval Health Research Center in San Diego and the National Cancer Institute's SEER database. The total naval population was used for comparison.

Tabulated were the numbers of men by diagnostic type of leukemia, totaling 102 verified cases out of 123 first hospitalizations for leukemia. In the acute-leukemia category, the larger incidences were for lymphoid (19 cases) and myeloid (18 cases). There were also 19 cases of chronic myeloid leukemia and

12 cases of unspecified myeloid leukemia; all other leukemia categories had 9 or fewer cases each. None of the SIRs for the various occupations that had three or more cases were significant relative to the SEER or to the total naval population except for "electrician's mate", which had SIRs and 95% lower confidence-interval limits exceeding 1.0. Men in this occupation operate, maintain, repair, and install electrical power plants, lighting systems, and other electrical equipment on ships, and they stand watch on shore near generators and other running electrical equipment.

The authors remarked: "This study, like other reported occupational studies of the possible association of [60-Hz] electric or magnetic fields and leukemia [citing McDowall (1983) and Tomenius (1986)] had no direct measure of exposure." As in Garland et al. (1987), some of the tabulated occupations that may have involved RFEMF exposure showed no significant excess leukemia incidence.

Garson et al. (1991) randomly selected 40 volunteers from about 250 radiolinemen who were employed by Telecom Australia to erect and maintain broadcasting, telecommunication, and satellite RFEMF transmission towers. The volunteers were matched with 40 unexposed clerical personnel by age and Australian State. Each exposed subject had worked at least five of the previous six years, and had been last exposed no more than 12 months before the study.

The exposure frequencies ranged from 400 kHz to 20 GHz. Before 1985, the exposure levels were within the ANSI/IEEE (1992) guidelines; afterward they were limited to the occupational levels of the 1985 Australian standard (AUS, 1985), which includes limits to avoid shock and burn. Specifically, the exposures of radiolinemen were limited to: 614 V/m and 1.63 A/m in the range from 400 kHz to 3 MHz, 61.4 V/m and 0.163 A/m between 30 MHz and 20 GHz, and intermediate frequency-dependent levels in the transition range 3-30 MHz. The authors indicated that in recent dosimetric measurements of radiolinemen at sites operating at up to 100 MHz, the induced current flow in each leg was less than the specified 100-mA limit.

Heparinized blood samples (10 ml each) were drawn from each exposed and control subject on the same day and treated in the same manner to minimize any chromosomal damage from handling or culturing. Duplicate cultures from each sample were prepared, and 100 metaphases from each culture (200 metaphases per subject) were counted and assessed for chromosomal damage in five categories: chromatid gaps, chromatid breaks, chromosome gaps, chromosome breaks, and "other" (more complex) abnormalities. The specimens were randomly coded, and were analyzed blind by only one of the authors, to minimize observer error. Of the 40 pairs of samples, two were discarded because one sample of each of those pairs did not produce metaphases, leaving 38 pairs for analysis.

The authors assumed that chromosomal damage occurs at a given rate and that any specific abnormality occurs independently of any other abnormality of the same or different type in the same subject. The Poisson distribution was used for statistical analysis of the data from the matched subjects. The results of the analysis are summarized in Table 18 (adapted from Table 2 of the paper). The authors noted that one of the **control** subjects exhibited at least 30 chromatid breaks, 14 chromosome breaks, and 3 other aberrations, totaling at least 47 aberrations. The data for that control subject were excluded as outliers, but were included in the table without 95% confidence intervals.

TABLE 18: CHROMOSOMAL DAMAGE IN RADIOLINEMEN
[Garson et al. (1991)]

Type of Abnormality	Rate per 100 Exposed Cells	Rate per 100 Control Cells	Rate Ratio (95% Confidence Interval)
Chromatid Gaps	0.54	0.46	1.2 (0.7-2.1)
Chromosome Gaps	0.21	0.14	1.5 (0.6-3.5)
Chromatid Breaks	0.20	0.61	
Without Outlier	0.18	0.22	0.8 (0.3-2.0)
Chromosome Breaks	0.95	0.88	
Without Outlier	0.97	0.72	1.4 (0.8-2.3)
Other Aberrations	0.63	0.64	1.0 (0.6-1.5)
Sum of Aberrations	2.53	2.74	
Without Outlier	3.55	2.18	1.2 (0.9-1.6)
Total Aberrant Cells	1.92	1.88	1.0 (0.8-1.3)

As seen in the table, most of the ratios of exposed-to-control rates slightly exceeded 1.0, but all of the 95% confidence intervals spanned 1.0, thus indicating that the differences between the exposed and control subjects were not significant ($p>0.05$). The authors remarked that smoking, recent infections, and X-rays were possible confounding factors, and therefore made adjustments for each such factor but not involving matched pairs (because of missing data on some subjects). They found that none of those factors materially altered the negative findings.

Garland et al. (1988b), in a brief report, discussed their study of the incidence of testicular cancer in active-duty enlisted personnel who had served in the Navy during 1974-1979. The histories and demographic data on those personnel were derived from the computerized database at the Naval Health Research Center (NHRC) in San Diego, California, comprising more than two million person-years. The study was conducted only on the white personnel because of the relatively low testicular-cancer incidence in the nonwhite personnel. The hospitalizations for testicular cancer during that period, numbering 143 cases, were identified in a computerized medical history file also maintained at the NHRC.

In Table 1 of the paper, the 143 cases were partitioned by age into several groups. The largest incidence for the Naval persons was in the age group 20-24 years: 67 cases comprising 964,189 person-years at risk and an average annual incidence rate of 6.9 cases per 100,000 person-years. However, the average annual incidence rate for that age group derived from National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) database for 1973-1977 was 9.3 cases per 100,000 person-years. The next largest incidence was in the age group 25-34 years: 48 cases comprising 597,022 person-years at risk and an average annual incidence rate of 8.0 compared to 9.3 cases per 100,000 person-years in the SEER database.

For the 143 cases overall, indirectly age-adjusted by reference to the SEER database, the average annual incidence rates were 3.7 and 3.9 cases per 100,000 person-years, respectively. Thus, no significant differences were found in age-adjusted or age-specific incidences of testicular cancer in the naval personnel. Although three of 110 occupations studied had elevated SIRs relative to SEER, those occupations involved daily exposure to a variety of chemical agents, many of which are known to be carcinogenic, but those occupations would not have involved much if any exposure to RFEMF. Thus, neither the negative nor the positive findings of this study provide any data about a possible link between RFEMF exposure and testicular cancer.

Hayes et al. (1990) did a case-control study of 271 individuals, age range 18-42 years, who were newly diagnosed with testicular cancer between 1 January 1976 and 30 June 1981, to determine occupational risk of testicular cancer relative to other types of cancer. The cases including 60 men with seminoma (a malignant neoplasm of the testis) and 206 with other germinal-cell tumors. Those cases had been referred for treatment to the Uniformed Services University Naval Hospital (USUNH), the Uniformed Services University Walter Reed Army Medical Center (WRAMC), or the National Institutes of Health

Clinical Center (NIHCC). Many of the cases were military personnel on active duty. The cases diagnosed prior to 1979 were ascertained from tumor registries, hospital admissions, and urology and pathology records at the three centers, and were interviewed by telephone; the cases diagnosed between 1979 and 1981 were interviewed in the respective hospitals.

The controls were 259 patients diagnosed at the same centers with cancer in other than the genital tract. Those diagnosed between 1976 and 1978 were identified from the computerized discharge logs at NIHCC and WRAMC and the tumor-registry records at USUNH, and were interviewed by telephone. Those diagnosed during 1979-1981 were identified on the oncology, surgical, and medical wards, and were interviewed in the respective hospitals. In addition, mothers of the case and control individuals were interviewed by telephone.

Controls were diagnosed during the same time period, and were frequency-matched to the cases by age ± 2 years. The authors noted: "To assess the possibility of biased results due to the use of cancer controls, some analyses were carried out limiting the control series either to subjects with lymphatic and haematopoietic malignancies or to subjects with other cancers." Of 335 cases with testicular cancer, 308 were alive, and interviews were completed for 271 of them, a response rate of 88%. Of 288 controls selected, 259 were interviewed, a response rate of 90%. The study population was 98% white, with a mean age at diagnosis of 27 years for both groups.

Of the interviewed testicular-cancer cases, 266 (98%) had germinal-cell carcinomas, grouped as seminomas (60 cases) and all other germinal-cell tumors (206 cases), with major histological groups classified as embryonal carcinoma (69) and teratocarcinoma (67). Of the five remaining cases, three had non-germinal (Leydig-cell) tumors and two had testicular tumors of unknown type. Of the controls, 29% had Hodgkin's disease; 18% non-Hodgkin's lymphoma; 17% melanoma; 7% each of soft-tissue sarcoma, bone tumors, and leukemia; 4% tumors of the nervous system; and 11% other cancers.

Information sought in the interviews included: job titles, activities and duties; materials handled; part-time or full-time status; name, type, and location of business; year started; and year ended. Also, the subjects were asked if they had been exposed to or handled: (1) radioisotopes, radioactive materials, nuclear materials; (2) radar equipment; (3) microwaves, microwave ovens, or other radio waves; (4) pesticides; or (5) polycyclic aromatic hydrocarbons. The authors stated: "Specific questions about exposure to electromagnetic waves were included to investigate an a priori clinical impression that radar and other microwave exposure were common among military testicular cancer cases."

The results were tabulated by occupation in terms of the odds ratios (ORs), with CIs (95% confidence intervals) and numbers of controls, for all testicular cancers combined, and by subdivision of the cases into seminomas and "other" germinal-cell carcinomas. The occupational titles shown were:

Professionals (administrator, teacher, physician/veterinarian)

Other professionals

Other white-collar workers (non-professional: health related, engineer/science, other technician, sales/service, clerk)

Blue-collar workers (agricultural/farming/fishing, construction, mining, transportation, mechanics/repair, garage service, electrical/repair, other repair, production, other manual)

Ship employment

Aircraft maintenance personnel.

Among the general findings were no significant excess risk for all testicular cancers, but a significant risk (OR=2.8, CI=1.4-5.4) for seminoma in the three professional subcategories above. Those in the

categories "other professionals" and "other white collar" were nonsignificant, and no increased risk for seminoma was associated with the blue-collar occupations.

Another general finding was for agricultural, forestry and fishery, and construction workers. The authors noted that the associated risks for testicular cancer were statistically significantly decreased, but did not provide a table of those results.

The ORs and CIs for the five categories of self-reported occupational exposures are displayed in Table 19 (adapted from Table 3 of the paper). Noteworthy were the results for the category "Microwave/other radiowaves": the ORs were significant for all testicular tumors, nonsignificant for seminomas, and significant for other germinal-cell carcinomas. On the other hand, the results for the category "Radar equipment" were nonsignificant for all three of the cancer classifications (all testicular tumors, seminomas, and other germinal-cell carcinomas). Not clear is why the authors did not regard "Microwave/other radiowaves" and "Radar equipment" as a single category.

TABLE 19: ODDS RATIOS* FOR TESTICULAR CANCER BY SELF-REPORTED EXPOSURE
[Hayes et al. (1990)]

		ALL TESTICULAR (N=271)		GERMINAL-CELL CARCINOMAS SEMINOMA (N=60)			OTHER (N=206)		
Exposure	No. of Controls	OR	CI†	OR	CI†	No.	OR	CI†	No.
Radioactive Materials	23	1.2	0.6-2.3	1.3	0.5-3.3	8	1.2	0.6-2.4	20
Radar Equipment	38	1.1	0.7-1.9	1.3	0.6-2.8	12	1.1	0.6-1.9	30
Microwave/ Other Radio	10	3.1	1.4-6.9	2.8	0.9-8.6	7	3.2	1.4-7.4	24
Hydrocarbons	12	1.5	0.7-3.4	1.0	0.2-3.9	3	1.7	0.7-4.0	15
Pesticides	29	1.2	0.4-1.2	0.1	0.0-1.0	1	1.5	0.9-2.7	32

*Odds ratios adjusted for age (16-21, 22-25, 26-29, 30-43)
†CI = 95% confidence interval

The authors did state that an independent assessment of self-reported exposures by job title did not support the significant elevations of risk for the "Microwave/other radiowaves" category. An industrial hygienist classified the exposures of the military patients to radar or other microwaves as: "high" (for electronic technicians and repairmen, power-generator operators and microwave-system operators), "medium" (for communications operators and signal officers), or "low" (for airplane flight crews). The results, displayed in Table 20 (adapted from Table 4 of the paper), indicate that the CIs for all of the ORs spanned 1.0 (nonsignificant). Moreover, the largest OR (2.3) was for those classified as having had low exposure, whereas the OR for the high-exposure group was only 0.8.

TABLE 20: ODDS RATIOS FOR TESTICULAR CANCER AMONG MILITARY PATIENTS BASED ON ASSESSMENT FROM JOB TITLE AND SELF-REPORTED EXPOSURE TO MICROWAVES AND OTHER RADIOWAVES
[Hayes et al. (1990)]

Exposure	None	Low	Medium	High	Any
Cases	116	10	6	12	28
Controls	107	4	6	14	24
OR	1.0	2.3	1.0	0.8	1.1
CI	----	0.6-9.4	0.3-3.8	0.3-2.0	0.6-2.1

Thus, the results of this study show no increase of risk of testicular cancer relative to other types of cancer from presumed occupational exposure to RFEMF, and provide no evidence that any of the types of cancer suffered by the patients was due to chronic RFEMF exposure.

Davis and Mostofi (1993), in a brief communication, reported hearing about six cases of testicular cancer that occurred between 1979 and 1991 among a cohort of 340 police officers employed at two police departments within contiguous counties (not identified) in the north-central U.S. The authors had interviewed the affected officers to collect information on their work environments; possible occupational exposures to RFEMF from radar guns; and past medical, occupational, and family histories.

The six cases had been employed as police officers as their primary lifetime occupation, and all had been exposed to traffic radar on a routine basis. Tabulated for each man were the year when hired, the year of first radar use, and the age and year of testicular-cancer diagnosis. The mean length of service prior to testicular-cancer diagnosis was 14.7 years, the mean age at diagnosis was 39 years, and all had used radar at least 4½ years before the diagnosis. Also noted were race (all were white) and other risk factors such as undescended testes, severe testes trauma, mumps orchitis, family history of testicular cancer, and occupational exposure to pesticides, herbicides, or dimethylformamide.

None of the six subjects was labeled as having had any of those risk factors, but one man was noted to have had mumps orchitis with atrophy in the noncancerous contralateral testis. Three of the six cases had embryonal cell carcinomas, one was diagnosed as having a mixed cell carcinoma with an embryonal cell metastasis, and the other two had seminomas. The authors stated: "The only common exposure shared by the officers was the occupational practice of resting the radar gun, while in the 'on' position, directly in the lap either close to, pointing at, or directly adjacent to the testicles."

Based on the interviews, the authors assumed that the size of the police departments was stable at 340 men. The authors used the 1981 SEER registry rates for white men 20-49 years of age and obscure reasoning to calculate the number of expected cases of testicular cancer in a cohort of men followed from 1963 through 1991. The result was 0.87 (O/E=6.9, $p<0.001$, Poisson distribution), but they did not provide any numerical data about the calculation.

The authors noted the following: "Disease clusters in the workplace historically have served to identify occupational exposures which cause disease [Fleming et al. (1991)]. However, cluster investigations, especially those of the general population, are notoriously difficult to interpret. Any time-space aggregate of disease can be defined in such a way as to produce an elevated cancer rate in any cluster [Rothman (1990)]." The authors then offered a brief but unclear discussion about how they avoided this pitfall. However, they also cited a review by the Electric Power Research Institute [EPRI, (1989)] of various studies, none of which found an association between exposures to electric and magnetic fields in the extremely-low-frequency (ELF) range and testicular cancer. On the other hand, they remarked: "Testicular cancer has previously been noted among microwave radar operators, but the significance of this report [Mostofi and Davis (1989)] is uncertain." [The latter citation was not included in their reference list, and could not be found in a search of several databases.

The findings reported in this communication provide no credible evidence that the incidence of testicular cancer in the 6 police officers was associated with their exposure to the RFEMF from the traffic radar guns they had used.

Fisher (1993) measured the X-Band and K-Band RFEMF levels emitted by 30 different models of police traffic radars from six manufacturers, including hand-held devices and those with antennas mounted in fixed configurations. The data were collected during calendar years 1982-1991. The total number of antennas evaluated was 5000 (1075 for X-Band and 3925 for K-Band).

"Aperture power densities" were measured, using a Narda Model 8621B isotropic probe that covers the frequency range from 300 MHz to 26 GHz, a Narda Model 8616 electromagnetic radiation monitor, and a Hewlett-Packard Model 3435 Digital Multimeter. The 8621B isotropic probe is independent of polarization; its sensors are at the center of a 5-cm-radius polystyrene sphere. The output of each

antenna was measured at its aperture, with the surface of the probe sphere in contact with the aperture, hence the term "aperture power density". The author noted that such measurements were in the near field of each antenna, and hence the readings may not be equal to the power densities in the plane of the aperture, but are useful for comparisons among different radar units and for relating the measurements to far-field values.

During the years 1982-1991 covered by this paper, about 95% of the radar units tested had fixed-mounted antennas, and the remaining 5% were hand-held devices, two categories expected to yield different exposure levels for the operators. The six manufacturers were coded as M1-M6, with the preponderance of devices produced by M1 (10 models, 2537 antennas tested), M2 (5 models, 1440 antennas tested), and M3 (12 models, 1017 antennas tested). The 30 different antenna models were assigned codes A1-A30 and their antennas were designated as F (fixed-mounted) or H (hand-held). The radars were labeled X or K [Band], with respective operational frequencies of 10.525 or 24.150 GHz.

Shown in Table V of the paper were the average output power densities and standard deviations for the individual manufacturers A1-A11, and for the remaining manufacturers, the latter lumped under "Other-K" and "Other-X" and comprising only about 5% of the units. The values ranged from 0.28 ± 0.03 mW/cm^2 for A5 to 3.36 ± 0.56 mW/cm^2 for A4 (both X-Band radars with fixed-mounted antennas). For A9, the only hand-held category listed (142 devices for K-Band), the average output power density was 0.44 ± 0.07 mW/cm^2.

In Table VI, data were presented as average, minimum, and maximum power densities for the fixed-mounted and hand-held antennas in the two bands. For the fixed-mounted X-Band antennas, the average value was 1.90 mW/cm^2, with minimum and maximum values of 0.1 mW/cm^2 and 6.4 mW/cm^2; the corresponding values for the hand-held X-Band antennas were 2.66, 0.3, and 4.0 mW/cm^2. For the fixed-mounted K-Band antennas, the average value was 0.93 mW/cm^2, with minimum and maximum values of 0.2 mW/cm^2 and 4.6 mW/cm^2; the corresponding values for the hand-held K-Band antennas were 0.69, 0.2, and 4.3 mW/cm^2.

The author cited NBS (1981), a report that described the free-space measurements on seven K-Band and 15 X-Band radar devices done by the National Bureau of Standards [now the NIST]. From those measurements, he formulated a simple model for predicting average power densities from measured aperture power densities for both fixed-mounted and hand-held X-Band and K-Band antennas at specific distances within the near- and far field of radars used for measuring speed.

He described three specific situations about the RFEMF levels to which the operators of such radars in a patrol vehicle may be exposed. These were: the region occupied by the operator when the antenna is fixed-mounted outside of the vehicle, behind and to the left of the operator; the operator's position when operating a hand-held radar; and operator's position when the antenna is fixed-mounted within the vehicle at the center of the dashboard, close to the front windshield. The author, citing exposure standards [ANSI (1982) and ANSI/IEEE (1991)], stated: "We are able to conclude with a high degree of certainty that there is no evidence to support the allegation that police traffic radar operators are at risk due to prolonged exposure to microwave emissions from their radar units."

Balzano et al. (1995), in response to claims of an association between testicular cancer and exposure to the RFEMF from hand-held traffic radars, described measurements of equivalent power density at the antenna aperture of a Kustom Signals Falcon 24-GHz hand-held traffic radar, and measurements of penetration depth and energy deposition in a material that had RF-absorption properties similar to those of human tissue. Noting that measurements previously done by others with use of electric-field (E-field) probes of large sizes relative to the local field variations in the plane of an aperture were likely in error, the authors used an EIT Model 979 E-field Probe and a NARDA/BRH Model 14 Implantable Probe. The sensors of the EIT probe are 2.5 mm long, or about 1/6 of the wavelength at 24.15 GHz (12.5 mm), and those of the NARDA probe are 1 mm long, or less than one-twelfth of that wavelength. Because of the sensitivity limitations related to the shorter length of the latter, the EIT probe was used for most of the measurements.

For calibration, the conical horn of the Falcon radar was modified by inserting a metal septum parallel to, and on the axis of the horn as a short-circuit in that plane, to obtain linear polarization. The authors described in detail the procedures used to calibrate the probes and to minimize errors from possible interactions of the transmission lines feeding the probes with the fields to be measured.

The major results were displayed in Figures 8 through 11 of the paper as two-dimensional plots of E^2 (the square of the E-field strength) within the face of the radar horn and in the planes at several distances from the horn face along the central propagation axis of the horn. As expected in the near-field (Fresnel) zone, the spatial plot of E^2 within the horn face (r=0) was a very complex pattern. The plot at r=5 cm was far less complex, but with a dip (crater) in the center of the pattern. At r=9.5 cm, the pattern was much smoother and without the central crater. A plot of on-axis E^2 values versus distance along the axis (Figure 12 of the paper) indicated that the far-field zone starts at about r=15 cm.

As noted by the authors, such complexity is explained by regarding the aperture of the horn as a set of tiny subapertures or elementary radiators, not necessarily in phase with the other subapertures, with each subaperture contributing to the total field at any measurement point. The summed fields at the small pathlengths within the plane of the aperture vary rapidly from point to point, giving rise to the complexity mentioned above.

At larger distances from the horn aperture, the spatial variations are progressively smaller than their corresponding distances to any measurement point, so the patterns are less complex than at r=0, such as for r=5 cm and r=9.5 cm. Thus, the energies associated with such rapid variations diminish with distance along the horn axis ("evanescent") and are absent in the far-field zone of an antenna. The authors noted that field measurements within the horn aperture or within planes close to the aperture include the energies of evanescent fields as well as those that propagate as radiation from the horn. For this reason, use of near-field measurements to derive the equivalent power densities propagating in the far field will overstate such power densities.

Forward, reflected, and radiated powers were measured with the radar horn radiating into free space and with the horn aperture held or pressed against various parts of the human body. The results showed that RF energy at 24 GHz is readily absorbed within the body, with only a small fraction reflected back.

The authors indicated that the thickness of human skin varies from about 0.3 mm to 2.0 mm, is rich in water, and that water is a good absorber at 24 GHz. They also remarked that ideally, the penetration depth should be measured with a small E-field probe implanted in tissue-equivalent material, but because the wavelength in tissue at 24 GHz is considerably smaller than in air, even the NARDA/BRH Model 14 implantable probe would be too large for such measurements. Instead, a chamois skin wetted with 20 g of water and stretched across the horn aperture served as a surrogate for human skin.

With a single 0.4-mm-thick layer of wetted chamois skin over the horn face, the distribution of E^2 was made within a plane at r=2 mm from the horn aperture, and similarly with a double layer of chamois. The results for the single layer showed substantial attenuation of the E^2 pattern measured previously in the absence of the chamois and even further attenuation with the double layer. By calculation, the attenuation of the chamois was 17 ± 2 dB/mm, corresponding to a penetration depth of about 0.5 mm, or about a third of that expected for a planewave in skin. The authors remarked that greater accuracy would be achieved if the surrogate skin were followed by a simulated fatty layer, but the error in the absence of such a layer was small because of the high attenuation in the surrogate skin.

In their conclusion, the authors indicated that for the 12-mW radar unit measured, the maximum power density incident on the skin when the antenna is in close proximity is less than 0.5 mW/cm^2, and that more than 95% of the energy is absorbed in the first millimeter of depth when the antenna is placed in contact with tissue. They also remarked that the penetration depths for infrared wavelengths are comparable to those at 24 GHz measured in this study, and found it interesting that the maximum allowable exposure from IR lasers is 100 mW/cm^2 (ANSI, 1993).

In overall conclusion about incidence of testicular cancer from RFEMF, including presumed exposure from traffic radars, based on analyses of the foregoing studies, there is no scientifically credible evidence to support such an association.

Schlehofer et al. (1990), citing a number of studies that reported an association of brain-tumor incidence with exposure to various chemical and physical agents (including ionizing and nonionizing electromagnetic fields), did a case-control study of occupational risk factors for brain tumors in a specific region of the Federal Republic of Germany for the years 1987-1988.. During those years, the region contained 1.3 million inhabitants, and brain-tumor cases were identified at two neurosurgical clinics in the region. Only patients with histologically confirmed primary tumors of the glial and meningeal tissues and of the acoustic nerve were selected. They comprised 99 males and 127 females residing in the region when diagnosed. Of those 226 cases, 51% were diagnosed with glial tumors, 56% with meningeal tumors, and 13% as "other" (mainly acoustic neuromas). The cases were interviewed in the hospital. For 10 patients who were unable to be interviewed, their next-of-kin were interviewed instead. The overall response rate was 97.8%. Controls, numbering 418, were randomly selected from the local residential registers, frequency-matched by age and gender to the case distribution, and interviewed.

The standard questionnaire used for both groups included details about possible environmental, life-style, and medical-risk factors. Data were also collected on residential history, occupation, smoking, previous diseases, drug consumption, and nutritional habits. The participants were categorized as nonsmokers (not more than 100 cigarettes during their lifetimes), current smokers, or exsmokers (quit at least five years prior to the interview).

A major finding was no elevated brain-tumor risk for smoking. More pertinent were the data for occupational exposure in 16 categories, one of which was "electricians", for 5 or more years. For both genders, there were 13 such cases (6%) and 14 controls (3%); the relative risk (RR) was 1.87 with a 95% confidence interval (CI) of 0.9-4.1 (nonsignificant). However, the 13 cases consisted of 6 females and 2 males with 5-20 years as electricians, and 2 females and 3 males with more than 20 years in that occupation. The only statistically significant finding was for the 6 females with 5-20 years as electricians: RR=6.22 (relative to 2 controls) and a CI of 1.2-31.7 (Table 5 of the paper). For the 2 females with more than 20 years, RR=4.56 and CI=0.4-31.9 (nonsignificant). The results for all 8 female cases were given as RR=5.2 and CI=1.4-20.1 (significant), but the authors recognized that the numbers of cases were too small to ascribe any credence to this single positive finding.

Maskarinec et al. (1994) noted an observation by a pediatric oncologist of an unusual number of children with leukemia that lived at the Waianae coast on Oahu, Hawaii. They investigated whether the incidences, derived from the Hawaii Tumor Registry during the period 1979-1990, could be ascribed to the presence of two low-frequency radio towers (frequencies not stated) in the region (probably at Lualualei). They defined a case as a child under 15 years diagnosed with acute leukemia during those years who had lived in three census tracts (96, 97, 98) in the region before diagnosis. Twelve children comprised the cases studied: one each during 1979, 1980, 1985, 1986, and 1990; two each during 1982 and 1984; and three in 1983. No cases were found during 1981, 1987, 1988, and 1989. The years with single cases were diagnosed as acute lymphocytic leukemia (ALL). The two 1982 cases consisted of one diagnosed as ALL and the other as nonlymphocytic leukemia (ANLL), as were the two cases in 1984. The three 1983 cases were all diagnosed as ANLL. Four sex- and age-matched controls who had resided in municipal Waianae at diagnosis time were selected for each case.

All addresses prior to the age of diagnosis were located on a map, and the distances fron the radio towers were estimated to within 0.2 mile both manually and by use of a geographical software package. The authors noted that this distance is the median of the distribution for all distances and that it also roughly delineates the valley where the radio towers are located.

Matched odds ratios were calculated, using the SAS (1989) software package. The standardized incidence ratio (SIR) was 2.09 for all 12 cases with a 95% confidence interval (CI) of 1.08-3.65 (significant), the SIR for ALL was 1.58 with a CI of 0.63-3.26 (nonsignificant), and the SIR was 3.73 for ANLL with a CI of 1.20-8.71 (significant). The authors remarked that the statistical power of the findings

was low because of the small number of cases, and they specifically stated: "It appears that closeness to the low-frequency radio towers has a weak association with leukemia, even though it was not statistically significant." They also noted that the Environmental Protection Agency (EPA) had measured the electric and magnetic fields around Lualualei Naval Station in 1990 and found that the levels did not exceed existing guidelines, citing EPA (1992). Thus, little if any credence can be given to the findings regarding an association of leukemia with exposure to the RFEMF from those towers.

Cantor et al. (1995) sought to determine whether a relationship exists between the incidence of female breast cancer and occupational exposure in the U.S. to various substances, ionizing radiation, and radiofrequency and microwave electromagnetic fields (REMF). The authors obtained more than 2.5 million mortality records that were coded for occupation from 24 states for the years 1984-1989. For 59,515 female decedents, breast cancer was listed as the underlying cause of death, of which 5,970 (10%) were black women. Four controls per case were randomly selected from all noncancer deaths and were frequency-matched in age, gender, and race. After excluding homemakers (comprising 24,148 white cases; 1,858 black cases; 110,067 white controls; and 9,041 black controls), there remained totals of 33,509 (29,397 white and 4,112 black) case women and 117,794 (102,955 white and 14,839 black) control women.

The authors displayed 31 potential workplace-exposure agents in Table 1 of the paper, and created a job-exposure matrix based on the professional judgment of an industrial hygienist, supplemented by NIOSH's Job Exposure Matrix (Sieber et al., 1991) and OSHA's Integrated Management Information System (Stewart and Rice, 1990). Some of the 31 categories were underlined to indicate that the matrix was used to estimate the odds ratios for "probability of exposure" on a scale 0 to 4, and "level of exposure" on a scale 0 to 3 for those categories. Categories 29 and 30 were assigned to radiofrequency and microwave electromagnetic fields, respectively, but only "radiofrequency electromagnetic fields" was underlined.

The odds ratios (ORs) of the white women for probability of exposure, displayed in Table 3 of the paper, were well below 1.2 for almost all of the agents listed, but for several ORs, the lower bound of the 95% confidence intervals (CIs) exceeded 1.00 (statistically significant). As an interesting example, the largest OR was for solder at exposure-probability 4: OR=2.97, with a CI of 1.3-6.6. (The latter was also the widest CI). However, there were only 11 cases and 19 controls. Similar results were shown for the black women in Table 5 of the paper: Again the OR for solder was highest but at exposure-probability 3: OR=2.40, with a CI of 1.3-4.5. [Results for exposure-probability 4 were not displayed for that category.]

More relevant in the present context are the results for the white women and the black women presumed exposed to radiofrequency electromagnetic fields and classified by exposure level (on a scale of 0-3), shown respectively in Tables 21 and 22 (adapted from Tables 4 and 6 of the paper).

TABLE 21: BREAST CANCER AMONG WHITE WOMEN BY EXPOSURE LEVEL
[Cantor et al. (1995)]

Exp. Cat.	Exp. Level	Cases	Controls	OR (1)*	OR (2)**	95% CI
REMF	0	24,505	84,484	1.00	1.00	--
REMF	1	1,183	4,260	0.96	1.15	1.1-1.2
REMF	2	1,940	6,756	0.99	0.85	0.8-1.0
REMF	3	1,369	5,734	0.82	1.14	1.1-1.2

*Adjusted only for age (at death).
**Adjusted for age and socioeconomic status (imputed from occupation).

TABLE 22: BREAST CANCER AMONG BLACK WOMEN BY EXPOSURE LEVEL
[Cantor et al. (1995)]

Exp. Cat.	Exp. Level	Cases	Controls	OR (1)*	OR (2)**	95% CI
REMF	0	3,437	12,596	1.00	1.00	--
REMF	1	160	487	1.13	1.23	1.0-1.5
REMF	2	243	815	1.09	1.02	0.8-1.2
REMF	3	236	772	1.12	1.34	1.1-1.5

*Adjusted only for age (at death).
**Adjusted for age and socioeconomic status (imputed from occupation).

For both the black women and the white women, the results for exposure-levels 1 and 3 were apparently significant, but the ORs barely exceeded 1.00 and the lower boundaries of the CIs were barely above 1.0. Moreover, the ORs of both groups for exposure-level 2 were nonsignificant, an indication of the absence of a relationship between of breast-cancer incidence and exposure level.

In their discussion, the authors indicated the many limitations of their study, not the least of which was the inability to control for most recognized breast-cancer risk factors. Specifically regarding ionizing and nonionizing radiation, they stated: "Ionizing radiation is known to cause breast cancer [citing Land et al. (1980)]. Nonionizing electromagnetic radiation has been hypothesized to also increase risk [citing Stevens (1987) on electric power use]; two studies of male breast cancer support this [citing Tynes and Anderson (1990) and Demers et al. (1991)]. In this investigation, we found no association with either ionizing or nonionizing radiation." In the last sentence, the authors rightly discounted the few statistically significant ORs for radiofrequency electromagnetic fields and the nonsignificant ORs for ionizing radiation.

In summary, this study provides neither positive nor negative scientific evidence of a relationship between exposure to RFEMF and incidence of female breast cancer.

Tynes et al. (1992) examined the risk of cancer in a cohort of 37,945 electrical workers in Norway 20-70 years of age, based on the job descriptions derived from the 1960 census. The International Standard of Classification of Occupations (ISCO) was used to classify the occupations. Listed in the paper were 12 occupations, two of which presumed exposure to RFEMF: "Radio/telegraph operators" (codes 676-677) and "Radio/television repairmen" (code 763). The numbers of persons in the two RFEMF-related subcohorts respectively were 1,641 (4.3%) and 1,376 (3.6%). The 10 other occupations presumed exposure to fields at powerline frequencies. The authors noted that the classifications were done a posteriori independently of the pattern of results, and that with no field measurements, the classifications may be somewhat inaccurate.

All of the 12 categories of electrical workers were linked by their personal identification numbers to a registry of cancer mortality and morbidity, which held the date of death or emigration and any details of cancer diagnosis. Each worker was under observation from the start of 1961 until death or the end of 1985. The incidence of cancer in the Norwegian male population who were occupationally active in 1960 was used to calculate the expected number of cancer cases in terms of the 5-year, age-specific incidence rates for each year from 1961 through 1985. Standardized incidence ratios (SIRs) and 95% confidence intervals (CIs) were determined, and the results were considered significant if the CIs did not include 1.00.

The cohort above was called "group I". A subcohort consisting of those who were still economically active in 1970 was also studied. An "economically active" worker was defined as one who had recorded a job description in the 1970 census. The latter cohort was called "group II".

Tabulated for both cohorts were the numbers of observed cases of 25 different sites of cancer (by International Classification of Diseases 104-204), the corresponding expected numbers of cases, and the SIRs and CIs. The CIs for seven of the 25 sites exceeded 1.00 (significant), and the results for all

cancers (3,806 observed cases or about 10% of the entire cohort) were an SIR of 1.06 and a CI of 1.03-1.09.

Tabulated separately for each group above were the results for leukemia, specified as "Acute" (lymphocytic, myeloid, other), "Chronic" (lymphocytic, myeloid), and "Other leukemia". In group I, the total number of leukemia cases was 107, and all of the CIs spanned 1.00 (nonsignificant). In group II however (74 cases), only the SIRs and CIs for myeloid were significant, with an SIR for all leukemia of 1.41 and a CI of 1.10-1.76. Also, in both groups I and II for the RFEMF-related subcohort "Radio/telegraph operators", the SIRs for leukemia were nonsignificant. The results for the other RFEMF-related subcohort "Radio/television repairmen" in group I were also nonsignificant, but were significant for group II (5 cases), with SIR = 3.18, CI = 1.03-7.43.

Similarly tabulated were the results for brain tumors in groups I and II. For the two RFEMF-related subcohorts in both groups, the SIRs and CIs indicated nonsignificance.

Combining their leukemia data in group II for the two RFEMF-related subcohorts (totaling 9 cases), the authors concluded that there was a significant excess risk of leukemia among workers in occupations with the potential for exposure to radiofrequency fields. In their discussion, however, they remarked that the term "electrical worker" is too vague to be a good marker for exposure to electromagnetic fields, and that the results should be interpreted with caution in the absence of field measurements in the selected jobs. They also indicated that potential exposure of the workers to solvents, polychlorinated biphenyls, and soldering fumes may explain the excess risks shown.

For the reasons indicated and the small percentages of cases relative to the entire cohort analyzed, little if any credence can be given to the findings of this study.

Tynes et al. (1996) performed a study of breast-cancer incidence in seagoing female radio and telegraph operators in Norway who potentially had been exposed to light at night, RFEMF at frequencies in the range 405 kHz to 25 MHz, and ELF (50 Hz). The authors' hypothesis was that such exposure may have caused breast cancer if it increased the susceptibility of the subjects to sex-hormone-related cancer by diminishing the production of melatonin by the pineal gland.

The authors derived data from a Norwegian Telecom cohort (TC) of 2,619 women who had been certified as radio and telegraph operators between 1920 and 1980, of which 98% had worked on Norwegian merchant ships. The median age at certification was 23 years. The cohort was followed from 1961 through 1991. Lost in follow-up were 41 women due to surname change and 103 women who had emigrated.

The TC subjects were linked by personal identification number to a Norwegian Cancer Registry (Pedersen and Magnus, 1959) to derive the incidence of female cancer in those operators. Also derived from that registry was an occupational cohort (OC) containing a subcohort of the TC plus those females in job titles other than radio and telegraph operators at sea (telephone operators, pursers, cooks, kitchen assistants, and office clerks). A third group, called the fertility cohort (FC), was derived from a registry at the Norwegian Central Bureau of Statistics that contained individual reproductive histories of 1.1 million women born between 1935 and 1969 who lived in Norway for some time after 1960. The authors used the FC to adjust for differences in fertility, regarded as a well-established risk factor for breast cancer (citing Kvåle et al., 1987).

Also performed was a nested case-control analysis of 50 cases of breast cancer, with 4-7 matched controls from the TC that were selected among the certified radio and telegraph operators alive at time of diagnosis of the cases. The controls were selected from those born before 1920 (± 5 years) and matched by birth year (± 1 year). The mean employment duration at sea was 3 years.

Spot measurements of the RF fields were made in the radio rooms of three Norwegian ships that still had old-fashioned transmitters (ITT types ST 1600A and ST 1610A) representative of those used in the last three decades. For those measurements, the transmitters were operated at maximum power. The

unmodulated transmitted power for telegraphy in the frequency range 410-535 kHz was 1.5 kW. The transmitted powers for both unmodulated and amplitude-modulated telephony were 400 W in the range 1.6-3.6 MHz and 1.5 kW in the range 3.6-25 MHz. A Holaday Industries Model HI-3002 isotropic broad-band field-strength meter with electric and magnetic probes was used. The meter was calibrated in a TEM cell (Crawford, 1974) at the National Institute of Occupational Health in Umeå, Sweden, before use. Correction factors for measurements below the specified frequency ranges of the meter extended the ranges down to 5 MHz for magnetic fields and 0.5 MHz for electric fields.

Exposure to the RF fields in the radio rooms was ascribed primarily to leakage from the unshielded feed lines between the antenna and transmitters. The rooms were large, and the radio officers were generally about 1-2 meters from the transmitters and feed lines. At the operator's desk, the levels of the electric field and magnetic field were below the detection levels of the instrument, which were about 20 V/m at all RF frequencies, and 0.05 A/m for frequencies above 3 MHz and 0.15 A/m below 3 MHz. The highest values, 1.4 kV/m and 7.5 A/m, were found close to the unshielded feed lines.

Measurements were also made of the ELF magnetic flux density (B) in two of the ships, with the transmitters active and off. The values of B varied from less than 0.002 μT to about 6 μT at the operator's position, with the highest level when the transmitter was active. The authors remarked: "In summary, the field levels at the operator's desk were comparable to those in normal working places in Norway, and the background level in the radio room was comparable to levels measured in Norwegian homes".

Detailed job histories were collected for the cases and controls from the TC. Shift work was categorized as 0, 1, 2, or 3 to indicate the extent of their presence in the radio room during the day and at night, the latter reflecting possible exposure to artificial light. Also, travel through time zones was categorized as 0 or 1.

In the cohort analysis of the TC and OC, the basic statistical unit used was the number of years each employee was followed up, starting from her date of first certification and concluding at the end of the follow-up period. Each year contributed by each member of the cohort was classified by age and calendar year, and the person-years of all were summed by age and by calendar year. The "standard incidence ratio" (SIR) of cancer cases was calculated, with the number of females in the national population as the basis. That basis was determined from the 5-year, age-specific incidence rates in the national female population, to yield the expected number of cancer cases for each year of the follow-up period. Also calculated were the two-sided 95% confidence limits (CIs) for an assumed Poisson distribution of incidences, with the results considered significant if the CIs did not include 1.0.

The follow-up period yielded 72,105 person-years, during which 140 new cancer cases were observed and tabulated. From Table 1 of the paper, the highest incidence was for breast cancer (50 women), with a SIR of 1.5 and a CI of 1.1-2.0 (significant). Next in decreasing order by cancer type were for cervical cancer (14 women, SIR: 1.0; CI: 0.6-1.7) and uterine cancer (12 women, SIR: 1.9; CI: 1.0-3.2). The 64 remaining cases were distributed among the various other types of cancer, with less than 10 women in each type and no significant SIRs. The collective SIR for all cancer types was 1.2 with a CI of 1.0-1.4. Of the 50 breast-cancer cases (Table 2 of the paper), 15 were younger than 45 years, with a nonsignificant SIR; 14 were in the 45-49 age range, with a SIR of 1.8 and a CI of 1.0-3.0; 13 were in the 50-54 age range, with a SIR of 2.5 and a CI of 1.3-4.3; and 8 were 55 years or older, with a nonsignificant SIR.

The nested case-control data were analyzed by logistic-regression models for matched sets. The variables consisted of shift work (0, 1, 2, or 3) and travel through time zones (0 or 1), each multiplied by the number of years and cumulated. The incidence of breast cancer for the women 50 years and older and those younger than 50 years were analyzed as two separate categories, based on considerations of premenopausal and postmenopausal incidence. The two categories were normalized to the median value for the exposed women. The results for each category were tabulated in terms of odds ratios (ORs) and CIs for breast cancer versus employment duration and versus cumulative shift work.

The ORs for the women under 50 years old (Tables 3 and 4 of the paper), both unadjusted and adjusted for employment duration and cumulative shift work, were nonsignificant. For the women 50

years or older (Table 5 of the paper), the highest unadjusted OR was 5.9, for those employed for durations between 3.2 and 14.6 years (150 cases with 46 controls), but the CI was nonsignificant: 0.7-47.7; also, the corresponding adjusted OR was 1.5, with a CI of 0.1-22.2.

Regarding shift work for the women 50 years or older, the highest unadjusted OR was 6.1, for those with cumulative values between 3.1 and 20.7 years (12 cases with 32 controls), and the CI was 1.5-24.2 (significant). When adjusted for cumulative shift work, however, the OR was 4.3 with a CI of 0.7-26.0 (nonsignificant). The authors stated that "a trend test for ordinal levels of exposure was performed by assigning scores 1, 2, and 3 to the three levels of exposure", but they did not clearly define those levels. From this test, they reported a significant trend (P=0.01) in cancer incidence with increasing cumulative shift work, categorized as "none", "low", and "high" for which the unadjusted ORs were respectively 1.0, 3.3, and 6.1. However, the corresponding adjusted ORs were 1.0, 3.2, and 4.3, and the significance level of the trend was stated to be P=0.13 (nonsignificant).

In an analysis of the women 50 years or older in terms of employment duration and shift work before age 30 years (Table 6 of the paper), the unadjusted ORs for shift work respectively were 1.0, 3.2, and 4.6 for the "none", "low", and "high" shift-work categories. The OR for the "high" category was significant (CI: 1.3-17.1). Adjustment of that OR also yielded 4.6 but the CI was 0.1-7.5 (nonsignificant). In addition, the aforementioned trend test was significant (P=0.02) for the unadjusted data but nonsignificant (P=0.06) for the adjusted data.

From the foregoing, it is difficult to understand why the authors regarded their results as indicating a significant relationship between breast-cancer incidence and occupational exposure to electromagnetic fields and/or artificial light during night work in the radio room. The latter was based on a vague speculative tie between such exposure and inhibition of melatonin production, said to be effective in reducing the growth of melanoma. In addition, the authors recognized the limitations of their dosimetry: "The cohorts studied had potential exposure to light at night, and the ELF and RF fields, but no historical exposure data were available. Measurements at the operators desk indicated levels of RF fields for all frequencies that were lower than the occupational guidelines recommended by The International Radiation Protection Association (IRPA) [1988]."

Hocking et al. (1996) investigated whether people in the vicinity of a triangle of three TV towers in northern Sydney, Australia, had a higher incidence of cancer and mortality from exposure to the RFEMF from those towers than in the general population. Since 1956, the towers have been used to broadcast three TV services; a fourth service was added in 1965. The channel frequencies ranged from 63 MHz to 215 MHz; the corresponding wavelengths ranged from about 5 meters to 1 meter, noted by the authors to be close to human [whole] body resonances for which absorption of RFEMF is highest.

The population consisted of 135,000 people in the three municipalities surrounding the towers (the "inner area", within a radius of roughly 4 km), and 450,000 people in six nearby municipalities (the "outer area") for comparison on the basis of decrease of RFEMF intensity from the towers by the inverse-square law. The mean height of the towers is 130 meters, they are 1.5 km apart, and their maximum power is directed at a vertical angle just below the horizon. The authors remarked that large antennas produce nulls (regions of low signal) close to such towers, but assumed that their design would ensure that the signals in those near areas would be at least 10% of maximum levels.

Using data obtained on the frequencies, powers, broadcast periods, and gain reduction versus angle below the horizon, the authors calculated the power density at several distances from the geographic center of the three towers. Up to 2 km, the distance from each tower to each specific location was used in the calculations; beyond 2 km, the RFEMF from the towers was assumed to emanate from their geographic center. The total power density at each location was taken to be the sum of the power densities for the four TV signals. The authors stated that the calculations did not account for ground reflections or reductions of signal strength by buildings, vegetation, or ground undulations.

The results were displayed (in Figure 2 of the paper) as a graph of power density on a logarithmic scale versus distance from the geographic center on a linear scale. Most of the points on the graph were

for distances up to 2 km. The points at each distance showed a large power-density spread, e.g., ranging at 1 km from about 0.6 $\mu W/cm^2$ to 8 $\mu W/cm^2$. The remaining points were single values decreasing at 3, 4, 8, and 12 km to 0.02 $\mu W/cm^2$. The authors noted that these calculated values are far lower than the 1990 Australian exposure standard (AUS, 1990), and that some measurements done near Tower 1 by the Commonwealth Department of Communications yielded values about fivefold lower than calculated.

Cancer data by age, sex, and residence at time of registry were derived from the New South Wales Cancer Registry in the municipalities of interest for the years 1972 to 1990, a registry that distinguished between incidence and mortality. The cancers of interest were leukemia and brain tumor (the latter especially in children).

The leukemia data are shown in Table 23 in terms of age groups, sex, years, and numbers of cases for the population in the outer area, and similarly in Table 24 for those in the inner area (both adapted from Box 2 of the paper).

TABLE 23: LEUKEMIA INCIDENCE FOR POPULATIONS IN THE OUTER AREA AROUND THREE TV ANTENNAS
[Hocking et al. (1996)]

Age (years) & Sex	Period	No. of Cases	Person Years (X Million)	Rate Per Million Person Years
0-14, M	1972-1978	21	3.96	5.3
	1979-1984	18	2.95	6.1
	1985-1990	15	2.70	5.6
0-14, F	1972-1978	20	3.73	5.4
	1979-1984	18	2.80	5.4
	1985-1990	9	2.57	3.5
15-69, M	1972-1978	98	10.56	9.3
	1979-1984	70	9.44	7.4
	1985-1990	89	9.78	9.1
15-69, F	1972-1978	48	10.89	4.4
	1979-1984	55	9.59	5.7
	1985-1990	45	9.82	4.6
70 and Over, M	1972-1978	45	0.84	53.8
	1979-1984	71	0.84	84.9
	1985-1990	64	0.96	65.1
70 and Over, F	1972-1978	63	1.55	40.7
	1979-1984	59	1.49	39.5
	1985-1990	61	1.65	38.9

TABLE 24: LEUKEMIA INCIDENCE FOR POPULATIONS IN THE INNER AREA AROUND THREE TV ANTENNAS
[Hocking et al. (1996)]

Age (years) & Sex	Period	No. of Cases	Person Years (X Million)	Rate Per Million Person Years
0-14, M	1972-1978	8	0.78	10.3
	1979-1984	6	0.59	10.1
	1985-1990	3	0.63	4.7
0-14, F	1972-1978	9	0.73	12.3
	1979-1984	2	0.57	3.5
	1985-1990	5	0.61	8.2
15-69, M	1972-1978	36	3.41	10.5
	1979-1984	27	2.90	9.3
	1985-1990	20	2.97	6.7
15-69, F	1972-1978	30	3.74	8.0
	1979-1984	21	3.15	6.7
	1985-1990	19	3.16	6.0
70 and Over, M	1972-1978	23	0.28	82.2
	1979-1984	27	0.26	103.3
	1985-1990	23	0.29	79.2
70 and Over, F	1972-1978	18	0.59	30.3
	1979-1984	29	0.53	54.4
	1985-1990	31	0.56	55.7

The data for the numbers of cases and person-years are the sums across the years for each age group and sex combination, with the person-years as the sum of the appropriate mid-year populations; the rates shown were obtained by dividing each number of cases by its person-year value. The rate-ratios (RRs) and 95% confidence intervals (CIs) for the total population in the inner area to those for the total population in the outer area are displayed in Table 25, which indicate the incidence and mortality for all leukemias and for specific types thereof. The results for the children (0-14 years of age) are similarly shown in Table 26 (with both tables adapted from Boxes 3 and 4 of the paper).

TABLE 25: RISK RATIO (RR) OF CANCER INCIDENCE AND MORTALITY FOR THE POPULATIONS AROUND ANTENNAS IN THE INNER AREA TO THE POPULATIONS IN THE OUTER AREA
[Hocking et al. (1996)]

INCIDENCE	ICD-9 CODE	RR (95% CI)	CASES
Brain Tumor	191	0.89 (0.71-1.11)	740
Lymphatic leukemia	204	1.32 (1.09-1.59)	536
Myeloid leukemia	205	1.09 (0.91-1.32)	563
Other leukemia	206-208	1.67 (1.12-2.49)	107
All leukemias	204-208	1.24 (1.09-1.40)	1206
MORTALITY			
Brain Tumor	191	0.82 (0.63-1.07)	606
Lymphatic leukemia	204	1.39 (1.00-1.92)	267
Myeloid leukemia	205	1.01 (0.82-1.24)	493
Other leukemia	206-208	1.57 (1.01-2.46)	87
All leukemias	204-208	1.17 (0.96-1.43)	847

TABLE 26: RISK RATIO (RR) OF CANCER INCIDENCE AND MORTALITY FOR CHILDREN (0-14 YEARS) IN THE INNER AREA AROUND ANTENNAS TO THOSE IN THE OUTER AREA
[Hocking et al. (1996)]

INCIDENCE	RR (95% CI)	CASES
Brain Tumor	1.10 (0.59-2.06)	64
Lymphatic leukemia	1.55 (1.00-2.41)	107
Myeloid leukemia	1.73 (0.62-4.81)	19
Other leukemia	1.65 (0.33-8.19)	8
All leukemias	1.58 (1.07-2.34)	134
MORTALITY		
Brain Tumor	0.73 (0.26-2.10)	30
Lymphatic leukemia	2.74 (1.42-5.27)	39
Myeloid leukemia	1.77 (0.47-6.69)	11
Other leukemia	1.45 (0.30-6.99)	9
All leukemias	2.32 (1.35-4.01)	59

As seen in Tables 25 and 26 and noted by the authors, no increase in brain-cancer incidence or mortality therefrom was found in either group: the CIs for incidence and mortality all spanned 1.0.

For total-leukemia incidence, however, Table 25 shows that both the RR and the CI in the total populations exceeded 1.0, but the CI for mortality therefrom spanned 1.0 (nonsignificant). Within the total-leukemia heading, both the incidence and mortality RRs for lymphatic, myeloid, and other leukemia exceeded 1.0, but the corresponding CIs for myeloid leukemia spanned 1.0 (nonsignificant). Table 26 for those of ages 0-14 years shows that the RRs and CIs for incidence and mortality of total leukemia were significant, and within total-leukemia heading, both the incidence and mortality were significant only for lymphatic leukemia.

The authors also compared the incidence and mortality of childhood cancer in the inner area with those for the [presumably childhood] population in the whole of New South Wales, and similarly for the children

in the outer area. The results are displayed in Table 27 (adapted from Box 5 of the paper). They indicate that both the incidence and mortality for childhood leukemia in the inner area were statistically significant, whereas they were not significant in the outer area, results taken as a possible link with exposure to the RFEMF from the TV towers.

TABLE 27: CANCER INCIDENCE AND MORTALITY FOR CHILDREN (0-14 YEARS) IN THE INNER AND OUTER AREAS RELATIVE TO THOSE IN THE WHOLE OF NEW SOUTH WALES
[Hocking et al. (1996)]

	OBSERVED CASES	EXPECTED CASES	SIR/SM[a]	CI[b]
		INNER AREA		
INCIDENCE				
Brain Tumor	12	9.1	1.3	0.7-2.3
Leukemia	33	18.6	1.8	1.2-2.5
MORTALITY				
Brain Tumor	4	4.1	1.0	0.3-2.5
Leukemia	19	7.9	2.4	1.4-3.7
		OUTER AREA		
INCIDENCE				
Brain Tumor	52	43.4	1.2	0.9-1.6
Leukemia	101	88.8	1.1	0.9-1.4
MORTALITY				
Brain Tumor	26	19.7	1.3	0.9-1.9
Leukemia	40	38.7	1.0	0.7-1.4

[a]SIR = Standardized Incidence Ratio; SMR = Standardized Mortality Ratio.
[b]CI = 95% Confidence Interval.

The primary conclusions of this study were that brain-cancer incidence and mortality were not increased due to residential proximity to the TV towers, but that a possible association was found between such proximity and increased incidence and mortality of childhood leukemia, presumed due to the RFEMF from the TV towers. The authors did mention various possible study biases and confounding factors that may have affected the findings. However, even if the statistically significant increase in leukemia in the inner area were accepted at face value, there is no clear evidence that the increase is related to the RFEMF from the towers. The power densities calculated by the authors are questionable because of the disparity between those values and the actual measured values, a point recognized by the authors in their discussion of the possible mechanisms thereof. Thus, no credence can be given to RFEMF as the basis for the positive findings of this study.

Szmigielski (1996) examined the entire population of personnel with military careers in Poland during the years 1971-1985 for cancer morbidity. During that period, the mean yearly count of military personnel was about 128,000. For each year, about 3,700 persons (3%) were selected on the basis of their service records and documented exposures at service posts as having been occupationally exposed to radiofrequencies (RF) and microwaves (MW).

Data on all newly diagnosed cases of malignant neoplasms were collected from the records of the central and regional military hospitals and central military board. The data on each person included first diagnosis of cancer, its localization and type, results of basic medical tests relative to the diagnosis, duration and type of service, exposure to possible carcinogenic factors during service, life habits, social/family status, and exposure to electromagnetic fields. Care was taken to avoid possible duplication of cases registered from different sources.

The author gathered data on exposure of personnel to RF/MW from military electromagnetic-field-safety groups that operated health hygienic services and that had responsibilities for measuring field intensities at and around service posts where RF/MW-emitting equipment was used and repaired, and for keeping personnel health records at those posts. The author stated: "The number of personnel considered to have been exposed occupationally to RF/MW was easily established, but evaluation of the exposure rate appeared to be quite difficult." He also indicated that at 80-85% of the posts, the RF/MW field intensities (mostly for pulse-modulated 150-3500 MHz) did not exceed 2 W/m^2 (0.2 mW/cm^2); at the other posts, the intensities were 2-6 W/m^2 (0.2-0.6 mW/cm^2), with frequent short-lasting exposures exceeding 6 W/m^2.

The career personnel in active service were divided into four 10-year age groups: 20-29, 30-39, 40-49, and 50-59 years, and data on morbidity (first malignancy diagnosis) were derived for each age group and for the whole population. The neoplasms were classified in terms of 12 occurrence sites, and the numbers of RF/MW-exposed (observed) and nonexposed (expected) incidences per 100,000 each year were averaged over the 15-year period. The author noted that the number of personnel occupationally exposed to RF/MW fluctuated annually from 3,400 to 4,600, with a mean of 3,720 ± 360. The expected rate of cancer morbidity was taken as the incidence for the total population, which consisted of more than 120,000 military-career subjects, including the RF/MW-exposed personnel.

Displayed in Table 1 of the paper were the [mean annualized] observed-to-expected ratios (OERs) of cancer incidence at the 12 sites, their 95% confidence intervals (CIs), and their probability (P) values without regard to age-group division. The OERs for 5 of the 12 sites were tagged as statistically significant: esophageal and stomach: OER=3.24, CI=1.85-5.06, P<0.01; colorectal: OER 3.19, CI=1.54-6.18, P<0.01; nervous system (including brain tumors): OER=1.91, CI=1.08-3.47, P<0.05; hematopoietic system and lymphatic organs: OER=6.31, CI=3.12-14.32, P<0.001, and skin (including melanomas): OER=1.67, CI=0.92-4.13. P for the last item was shown as <0.05, but the CI spanned 1.0 and therefore was not significant. The OERs for the remaining 7 sites (oral cavity, pharynx, liver and pancreas, larynx and lungs, bones, kidneys and prostate, and thyroid) were not significant (P>0.05). All observed malignancies per 100,000 totaled 119.12 persons versus 57.60 expected, for a ratio of 2.07 and a CI of 1.12-3.58 (P<0.05).

In the results above, the largest OER and the highest probability was for cancers of the hematopoietic system and lymphatic organs. The results for the seven subtypes under this category were displayed in Table 2 of the paper. In decreasing order of OER, they were:13.90 for chronic myelocytic leukemia, CI=6.72-22.12, P<0.001; 8.62 for acute myeloblastic leukemia, CI=3.54-13.67, P<0.001; 5.82 for lymphoma (non-Hodgkins) and lymphosarcoma, CI=2.11-9.74, P<0.001; 5.75 for acute lymphoblastic leukemia, CI=1.22-18.16, P<0.05; 3.68 for chronic lymphocytic leukemia, CI=1.45-5.18, P<0.01; 2.96 for malignant lymphogranulomatosis (Hodgkin's disease), CI=1.32-4.37, P<0.05; and 2.12 cases of myeloma (plasmocytoma) for the exposed group versus none expected (unexposed) cases per 100,000, P undetermined. Overall, the OER was 6.31, CI=3.12-14.32, P<0.001.

Regarding the results for chronic myelocytic leukemia (the largest OER), the incidences were 12.23 cases per 100,000 (0.012%) for the exposed group versus 0.88 cases per 100,000 (0.001%) expected cases, yielding the 13.90 risk ratio above. The incidences for all types of hematopoietic/lymphatic malignancies were 43.12 exposed cases per 100,000 (0.04%) versus 6.83 expected cases per 100,000 (0.007%), yielding the 6.31 overall risk ratio.

The author noted that the size of the population studied varied from year to year, with a minimum of 118,500, a maximum of 142,000, and a mean of 127,000 ± 9,620 (standard deviation) for the 15-year period. Also noted was that the exact age distribution is still classified, so the age-distribution results could be given only as incidence rates and odds ratios, as shown in Table 3 of the paper. For all neoplasms, the OERs in decreasing order were: 2.33 for the 20-29 age group, CI=1.32-3.12, P<0.05; 2.30 for the 30-39 group, CI=1.04-3.06, P<0.05. For the 40-49 group, the OER was 1.92, CI=0.98-2.84, labeled significant P<0.05) but from the CI was not; and the OER for the 50-59 group was 1.47, CI=0.92-2.12, P>0.05 (nonsignificant). Specifically for the hematopoietic system and lymphatic organs, the OERs

for the 20-29, 30-39, 40-49, and 50-59 age groups respectively were 8.16, 8.58, 8.80, and 4.47, all significant ($P<0.01$). The overall OER for all age groups was 6.31, CI=3.12-14.31, $P<0.001$.

The author stated: "At present, it is not possible to offer a reasonable explanation for the threefold increase of the rate of stomach and colorectal adenocarcinoma in RF/MW-exposed military personnel in Poland or to be certain about causal links with the exposures. Nevertheless, we were not able to find any differences in dietetic or life habits, consumption of alcoholic beverages or possible exposure to other occupational carcinogenic substances that may explain the differences in morbidity between the exposed and non-exposed servicemen."

To support the foregoing, the author did not present statistical data on any of such comparisons between RF/MW-exposed and nonexposed personnel on their life habits, social/family status, or other such possible confounding factors. Not clear was how the numbers of unexposed personnel that comprised the expected numbers used in calculating the OERs were derived. A presentation of the actual numbers of exposed cases and unexposed controls each year rather than data in the form of the averaged annual numbers of cancer cases per 100,000 over the 15-year period would have provided a better basis for assessing the robustness of the findings, particularly in view of the uncertainties noted by the author about the levels and durations of RF/MW exposure. Not discussed were the specific kinds of duties performed by the personnel at RF/MW posts that would have distinguished those at potentially greater risk of occupational exposure from those for non-RF/MW personnel at the same posts. Regarding the results indicating progressively lower OERs with increasing age group (Table 3 of the paper), they are difficult to interpret because of the lack of any information on the ages of entering military service and the durations of their tours of duty.

Grayson (1996) did a nested case-control study of possible brain-tumor risk from presumed exposure to non-ionizing and ionizing radiation of a group within a cohort of 880,000 Air-Force personnel who had at least one year of service during the years 1979-1989. The cases were ascertained by screening Air-Force hospital discharge records of men who had served during that period and had been diagnosed for primary malignant brain tumor. Also included were those who had been treated at other than Air-Force facilities. Of 246 cases identified, 16 were eliminated because of incomplete or conflicting personnel data, yielding 230 cases.

Selected from the cohort for each case were four controls matched for year of birth and race, and who were part of the cohort when the case was diagnosed. Not eligible as controls were those who had been diagnosed with leukemia, breast cancer, or malignant melanoma.

The author provided separate estimates of case exposures to extremely-low-frequency (ELF) fields, RFEMF, and ionizing radiation. The estimates for exposure to RFEMF were derived from a central registry of all incidents of Air-Force personnel who were reported to have been exposed at levels that exceeded 10 mW/cm^2 [the frequency-independent maximum permissible power density in ANSI (1974)] since 1972. Based on that Air-Force registry, he developed a job-exposure matrix that categorized Air-Force job titles over time as having had "no", "possible", or "probable potential" for exposure to RFEMF.

"Probable intensity" scores were assigned to occupations in which the overexposures had been reported, as well as to closely related job titles. As noted by the author, this included all occupations involved in the maintenance and repair of RFEMF emitters. The "possible intensity" score was assigned to those occupations requiring operation of RFEMF emitters for which excessive exposures had not been reported. All other job titles were assigned to the "non-exposed" category. However, the author did not provide a list of the specific occupational job titles, the scores for those titles, and the number of persons in each.

For each subject, estimates of his cumulative exposures to RFEMF and ELF were made by multiplying the exposure score by the number of months in each job title held during his career, and by summing those products. For exposure to RFEMF, 30% of the subjects were said to have had cumulative scores that ranged from 8 to 610 intensity-months. For ELF exposure, 48% had cumulative scores from 1 to 885 intensity-months. Again, no data were presented. By using conditional logistic regression models, the

author derived age-race-adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for brain tumors in the exposed-subject categories (for both RFEMF and ELF) relative to those in the non-exposed category and tabulated the results by the military-rank categories indicated below.

In Table 2 of the paper, the numbers of enlisted personnel in the ranks "airmen", "non-commissioned officers", and "senior non-commissioned officers" totaled 169 cases and 782 age-race-matched controls. The commissioned officers were categorized by rank as "company-grade officers" [second lieutenant to captain], "field-grade officers" [major to colonel], and "general officers" [brigadier general to lieutenant general], totaling 61 cases and 138 controls. The ORs for each rank category had CIs that spanned 1.0 (nonsignificant), but comparison of all officers as a group with all enlisted men as a group (Table 3 of the paper) yielded an OR of 2.11 with a CI of 1.48-3.01 (significant). Moreover, by grouping the field-grade and general officers together as "senior officers" (37 total) and comparing that group with the total of all others (193 enlisted men and company-grade officers), an OR of 3.30 and a CI of 1.99-5.45 (also significant) was obtained.

The findings of this paper are questionable for various reasons. First, the absence of data on the various occupational titles provides no basis for analyzing the validity of the results reported. Specifically, for example, not indicated were the scores assigned to the three exposure categories in the job matrix. Second, the use of the Air-Force registry of "overexposed" people to develop the exposure job matrix is questionable because each such incident presumably has been investigated to determine whether the person actually had been exposed, and if so, for how long and for any health consequences. Also, such incidents are rare and likely to occur in a small subset of occupational titles.

In the findings by military rank presented in the paper, it is surprising that the ORs for all commissioned officers and particularly those in the "senior-officer" category (as defined by the author) were significantly excessive relative to those in the enlisted category. The latter men were said to be far more likely to have been required to operate and/or repair equipment that emitted non-ionizing electromagnetic fields than the senior officers or the officers of lower rank.

For the reasons above, the findings of this study have no scientifically credible validity.

Grayson and Lyons (1996) studied the incidence of cancer in U.S. Air Force personnel who were pilots or had flown as crew during the period from 1975-1979. The comparison groups used were non-flying Air Force officers and the National Cancer Institute's Surveillance Epidemiology and End Results (SEER) program. Tabulated were the incidences of cancers of various types and body sites.

For all cancer sites, the Standardized Incidence Ratio (SIR) was 1.19 with a 99% confidence interval (99CI) of 1.03-1.36 for the air crews relative to the SEER group, and the risk ratio (RR) was 1.31 with a 99CI of 1.11-1.54 relative to non-flying officers. Contributing to the significant SIR were an excess of skin cancer with a SIR of 1.62 and a 99CI of 1.20-2.13 (primarily malignant melanoma) and of cancers of the urinary system with a SIR of 2.03 and a 99CI of 1.19-3.22. The SIRs for the remaining cancer sites listed were nonsignificant (99CIs that spanned 1.0), except for a paucity of Hodgkin's disease, with a SIR of 0.51 and a 99CI of 0.23-0.99. The risk ratios were nonsignificant except for genital cancers, with an RR of 1.69 and a 99CI of 1.12-2.56, and for cancers of the urinary system, with an RR of 2.33 and a 99CI of 1.27-4.27.

The authors did not discuss the influence of any recognized carcinogenic agents or those presumed to be potentially carcinogenic (such as RFEMF) that may have contributed to the few cancer excesses tabulated. Thus, the findings of this study provide neither positive nor negative evidence for a relationship between the incidence of cancer and exposure to non-ionizing electromagnetic fields.

The possibility that workers in occupations that involve the use of RFEMF for welding or sealing plastics would be exposed to excessive RFEMF levels was examined in several studies, among them Cox et al. (1982), Joyner and Bangay (1986), and Bini et al. (1986). Lagorio et al. (1997) examined in detail a cohort of factory employees in Italy who performed heat sealing with RFEMF. These studies are discussed below.

Cox et al. (1982) measured the electric and magnetic fields in the frequency range 18-31 MHz at the stations of 82 operators of dielectric heat sealers in 13 U.S. facilities. The authors found that 55% of those operators were being exposed to levels exceeding the maxima permitted in the OSHA (1978) exposure guidelines [respectively 200 V/m and 0.5 A/m, or 10 mW/cm^2 equivalent plane-wave power density, as in the ANSI (1974) guidelines]. However, Cox et al. (1982) did not discuss any specific health problems of those workers.

Joyner and Bangay (1986) did a survey of the electric and magnetic fields of 101 dielectric heaters operating at a frequency of 13.56, 27.12, or 40.68 MHz in Australia during 1982 and 1983. Their results showed that 39% of the heaters were exposing the operators to levels that exceeded the Australian guidelines by factors of more than 1 and less than 10, and that the levels of another 23% of the heaters exceeded those guidelines by factors greater than 10. The authors did not discuss possible health effects on the operators of those heaters.

Bini et al. (1986) described measurements of the RFR levels in a room (presumably in Florence, Italy) that contained 67 sealers used to make thermal seams in various plastic-sheet articles, such as inflatable boats. Most of the sealers operated at 27.12 MHz, with deviations of several MHz, depending on load conditions and type of applicator; a few of the other sealers operated at 13.56 MHz. The authors found that the electric field near most of the units exceeded the levels permitted in Italian exposure guidelines. However, they noted that the stray fields were essentially confined to the immediate vicinity of the units, so the hands of the operators received the highest exposures, followed by the head and the abdomen.

The authors briefly mentioned that 63 female workers were interviewed for possible health effects; of those, 30 had been exposed, 11 had been partially exposed (those not directly or continuously working at the sealers), and 22 had been unexposed controls. The exposed group reported subjective complaints of eye irritation and upper-limb paresthesia (abnormal sensations such as burning, prickling), with statistically significant incidence relative to the other groups. The laboratory tests given all three groups indicated no compromise of their general health. Clinical examinations of specific organs and systems of the exposed group did not yield evidence that the paresthesias were of neurogenic origin, but "vitreous-body disorganization" in various degrees was observed. The authors cited Desideri et al. (1986) [as in press] for a more detailed description (in Italian) of those health findings.

Lagorio et al. (1997) examined a cohort of workers in a factory in Grosseto, Italy, who were employed to perform dielectric-heat-sealing with RFEMF in the manufacture of plastic-ware products such as dinghies, lifeboats, and a few polyvinyl chloride (PVC) products. The building area used for such activities was a metal-shielded illuminated shed of area 400 m^2. Eligible for the cohort were 200 men and 481 women employed in the factory from 1 October 1962 or subsequently hired through 30 September 1992, based on payroll records. Personnel files were used to obtain information on personal data, dates of hire and employment termination, and periods of assignment to specific departments and jobs. Three subcohorts were defined:

RF-sealer operators consisting of 302 women and 4 men, totaling 6772 woman-years and 104 man-years at risk.

Other laborers consisting of 150 women and 114 men, totaling 3351 woman-years and 1808 man-years.

White-collar workers consisting of 29 women and 83 men, totaling 486 woman-years and 1021 man-years.

The authors stated that quantitative RF-exposure assessments were not attainable because of the high turnover and varying characteristics of the RF machines and the lack of information about assignments over time to different machines. Instead, they relied on the findings of an earlier survey [Bini et al. (1986)], when no metal shielding or grounding of the machines had been used. That survey showed that the maximum allowable exposure limit of INIRC (1988a), primarily the electric field strength,

had been exceeded frequently in this plant. [Note: The findings of Bini et al. (1986) were discussed in Heynick and Polson (1996b). No evidence of RF-induced cancer incidence was reported in RF-sealer operators.] The authors also noted that exposure of the workers to solvents in the sealing department was never monitored even though some vapors from the nearby (non-sealer) shed was foreseeable. In addition, the workers were exposed to vinyl chloride monomer (VCM) by volatilization from the PVC sheets during sealing; the level of VCM measured in 1983 was 37 ± 33 (SD) $\mu g/m^3$.

The Registry Office of the municipality in which each worker lived was used to ascertain whether that worker was alive and if not, the cause of death. For calculating person-years at risk of dying, each subject was considered at risk from 1 October 1962 (start date of the study) or date of hire if later until 31 December 1992 (end date of the study) or date of death if earlier. The observed numbers of deaths (O) from various causes of the RF-sealer operators were compared to the expected numbers of deaths (E) by use of the standardized mortality ratios (SMRs) and their 95% confidence intervals (CIs). The E values were determined by applying the gender, age, and calender period of the person-years at risk of dying to the regional mortality rates. The results were displayed in Table 2 of the paper for the female RF-sealer operators, the other female workers, and for all female workers, along with the corresponding SMRs and CIs.

Of 302 female RF-sealer operators with a total of 6772 woman-years at risk of dying, 9 (3%) had died from all causes. Of those 9 decedents, 6 had died from "Malignant Neoplasms" [ICD codes 140-208]: 1 each for a malignancy in the oral cavity or pharynx, liver, lung, uterus, brain, or from leukemia versus a total of 3 expected deaths from those causes. The SMR was 2.0 but the CI was 0.7-4.3 (nonsignificant). One of the remaining 3 had died from "Benign and unspecified neoplasms" (ICD codes 210-219) versus 0.08 expected (SMR and CI not displayed), and the other 2 had perished from accidents or violence (codes 800-999) versus 0.8 expected, with an SMR of 2.4 and a CI of 0.3-8.7. None of the female RF-sealer operators had died from circulatory or respiratory disease (codes 309-519) versus respectively 1.1 and 0.2 expected. The overall SMR for the female RF-sealer operators was 1.4 with a CI of 0.7-2.7 (nonsignificant). In the female other-workers group, the only significant SMR was for accidents and violence: 3 observed versus 0.4 expected, with an SMR of 7.0 and a CI of 1.4-20.5.

No results were presented in Table 2 of the paper for the male RF-sealer operators, presumably because the subcohort had only 4 men with a total of 104 man-years at risk of dying.

The authors concluded: "Although based on small numbers, we observed an increased risk of malignant neoplasms (seven cases), particularly leukemia (two cases). The majority of cancer deaths was concentrated among RF-exposed workers." No credence can be given to this conclusion not only because of the paucity of cases noted, but also the lack of adequate dosimetric data and the likely exposure of the RF-sealer workers to human chemical carcinogens, points recognized by the authors in their discussion. Moreover, the SMR for the excess of cancer deaths was nonsignificant, as indicated by a 95% confidence interval that spanned 1.0. Last, the malignant neoplasms in 1 decedent each (a total 6 decedents) occurred in various sites within the body, possibly indicating that the etiology of each cancer differed from the others.

Dolk et al. (1997a), in one of two studies, examined the incidences of various forms of cancer during the years 1974-1986 in the region around the West Midlands, England. The studies were undertaken because of an unconfirmed report of a cluster of leukemias and lymphomas near the Sutton Coldfield broadcast transmitters, located at the northern edge of Birmingham. (That report was summarized in the paper's Appendix.)

The authors noted that the Sutton Coldfield facility first started television broadcasting in 1949. Broadcasting at 1 MW erp per frequency was begun at one frequency in 1964, at three frequencies in 1969, and at four frequencies in 1982. Radio broadcasting at three VHF frequencies began in 1957 at 250 kW erp per frequency. The authors noted that the antenna mast is 240 meters high, there are no big hills higher than the mast in the area, and industrial processes nearby include a mineral works 3 km east, a copper works 6.5 km west, and a lead works 7 km west.

The geographic area considered was a circle 10 km in radius, with the transmitter site at its center; the area was divided into 10 bands of outer radii 0.5, 1, 2, 3, 4.9, 6.3, 7.4, 8.3, 9.2, and 10 km, to yield equal areas beyond 3 km. The population within the study area was about 408,000, derived from the 1981 census. Data on cancer incidence were gathered for the years 1974-1986 based on the postcode of residence of each case at diagnosis time. Childhood cancer (for ages 0-14 years) was restricted to "all cancers" and "all leukemias". For those diagnosed at age 15 years or older, the various forms of cancer were assigned a priori to the four groups below, each group including one or more codes in the 8th and 9th revisions of the International Classification of Diseases (ICDs):

1) All cancers, excluding non-melanoma skin cancer.

2) Cancers of the various types stated in the initial cluster report.

3) Cancers possibly associated with non-ionizing radiation [based on a number of specific papers cited by the authors].

4) Common cancers (examined separately).

To account for possible socioeconomic confounding, a deprivation score was calculated for each census enumeration district, based on the 1981 census data on unemployment, overcrowding, and social class of the head of household. Those scores, which were grouped into quintiles, indicated that areas closer to the transmitter were more affluent than those further away: within 1-2 km [the three nearest bands], 67% of the population was in the two most affluent quintiles versus only 28% within 9.2-10 km. The authors stated: "For many cancers (e.g. lung), lower incidence rates would be expected in the more affluent areas; for some other cancers (e.g. leukemia), there is essentially no relation between incidence and deprivation thus measured, whereas for others (e.g. skin melanoma), higher disease rates are found in the more affluent areas."

Calculated were the ratios of the numbers of observed cancer cases to expected cases (O/Es), with the expected cases determined from the national incidence rates stratified by 5-year age group, sex, year, and deprivation quintile. The authors noted that the West Midlands region had standardized O/Es of 0.95 for all cancers and 0.80 for leukemias (with an O/E of 0.65 for chronic lymphatic leukemia). They displayed the results for the people 15 years and older in more detail in Table 1 of the paper, in terms of the O/Es and their 95% confidence intervals (CIs) for both the entire 10-km area and the inner 2-km area around the transmitter, the latter arbitrarily chosen for comparison.

For the inner 2-km area, the numbers of observed and expected cases for "all cancers [except non-melanoma skin cancer]" were respectively 703 and 647.49, yielding O/E=1.09 and CI=1.01-1.17 (statistically significant). The CIs for "hematopoietic and lymphatic cancers" (45 observed and 37.08 expected cases) and for "all leukemias and non-Hodgkin's lymphomas" both spanned 1.0 (nonsignificant).

When the data for "all leukemias and non-Hodgkin's lymphomas" [within the inner 2-km area] were separated, those for "all leukemias" (23 observed cases and 12.59 expected cases) were significant, with "chronic lymphatic" (8 observed cases and 3.12 expected cases) the only significant subcategory. The results for "non-Hodgkin's lymphomas" (96 observed cases and 290.5 expected cases) were not significant.

Those for "multiple myeloma" (the last category in that table) were also non-significant.

For the entire 10-km area (17,409 observed cases and 16,861.22 expected cases), the results for "all cancers" and for "all leukemias and non-Hodgkin's lymphomas" (661 observed and 593 expected cases) were both significant. For the "all leukemias" subcategory, only "chronic lymphatic" (96 observed and 73 expected cases) was significant. The result for the "non-Hodgkin's lymphomas" subcategory (357 observed and 290.5 expected cases) was significant, whereas the corresponding result for the inner 0-2 km region was not significant, a result indicating no decline of risk with distance from the transmitter.

The authors interpreted the significant "all leukemias" results above (O/E=1.83, CI=1.22-2.74) for the 0-2 km range and the non-significant results for the 1-10 km range (O/E=1.01, CI=0.90-1.13) as indicating possible decline in risk with distance from the transmission site.

In more detail about the observed and expected incidences within each distance band, Table 28 (adapted from Table 2 of the paper) displays the O/Es for "all cancers", "all leukemias", and "non-Hodgkin's lymphomas" within each band area, but without the corresponding CIs. Not shown are the cumulative O/Es obtained by dividing the sum of the observed cases within the total area of the bands at each distance by the sum of the expected cases within that area.

TABLE 28: ALL CANCERS, ALL LEUKEMIAS, AND NON-HODGKIN'S LYMPHOMAS VERSUS DISTANCE BANDS
[Dolk et al. (1997a)]

	ALL CANCERS*			ALL LEUKEMIAS			NON-HODGKIN'S LYMPHOMAS		
Dist. (km)	Obs. (O)	Exp. (E)	O/E	Obs. (O)	Exp. (E)	O/E	Obs. (O)	Exp. (E)	O/E
0-0.5	2	5.61	0.36	1	0.11	9.09	0	0	0
0.5-1.0	96	137.19	0.70	5	2.72	1.84	3	2.60	1.15
1.0-2.0	605	504.59	1.20	17	9.76	1.74	5	9.46	0.53
2.0-3.0	282	279.01	1.01	9	5.56	1.62	9	5.76	1.56
3.0-4.9	1002	1050.56	0.95	25	20.22	1.24	20	20.25	0.99
4.9-6.3	2414	2301.25	1.05	54	41.96	1.29	45	40.60	1.11
6.3-7.4	2734	2650.62	1.03	48	46.54	1.03	57	43.95	1.30
7.4-8.3	2827	2798.65	1.01	51	49.22	1.04	52	47.19	1.10
8.3-9.2	3363	3213.75	1.05	40	57.35	0.70	80	54.56	1.47
9.2-10	4084	3919.59	1.04	54	68.90	0.78	86	66.02	1.30

*Excluding non-melanoma skin cancer.

As seen in the table, the O/Es for "all cancers" were close to 1.0, but the values for "all leukemias" appeared to indicate a decreasing trend with distance. Within the arbitrarily chosen 2-km distance from the site, however, the cumulative O/E (as defined above) was 1.83, but comprised only 23 observed cases versus 12.59 expected cases over the period 1974-1986. Also, the highest cumulative O/E was 2.12, but for only 6 observed cases and 2.83 expected cases.

The values for "non-Hodgkin's lymphoma" varied non-monotonically with distance, with the highest cumulative O/Es at the longest distances, a possible trend with distance opposite to that for "all leukemias".

Overall for the period 1974-1986, the total number of observed leukemia cases within the 10-km region was 304 persons, which when divided by the population in that region taken from the 1981 census (about 408,000 people), comprised about 0.07%. By contrast, the number of observed "all cancer" cases was 17,409 or about 4% of that population, but the results for that inclusive category were nonsignificant.

The use of the population in 1981 (midpoint year of the period covered) for normalizing was one of several possible methodological artifacts mentioned by the authors. They noted that estimates of the population changes based on data from the 1971 and 1991 censuses may have yielded overestimates of the O/Es near the site, but that the bias (estimated as less than 5%) was not sufficient to explain the excessive leukemia found. Unclear is the influence (if any) of the deprivation score. The industrial processing plants located in the region were not discussed as a possible confounding factor. The authors noted measurements of power density by the British Broadcasting Corporation in 1994, but also recognized the great variability of those data and the lack of data on the levels and exposure durations of the people involved, a problem common to most such epidemiologic studies.

For the reasons above, it is difficult to ascribe any credence to the findings of this study.

In the second study, Dolk et al. (1997b) investigated cancer incidences near the 20 other high-power FM and TV transmitter sites in Great Britain for the period considered in the analysis of the Sutton Coldfield site in Dolk et al. (1997a)], to take advantage of the much larger population samples. All 21 sites were grouped by their effective radiated powers: Group 1 comprised those transmitting TV with an erp in the range 870-1,000 kW. Group 2 consisted of the sites transmitting TV with erps of 500-1,000 kW. Group 3 included all FM sites transmitting at 250 kW erp. Group 4 comprised all sites emitting both TV (at 500 kW erp) and FM (at 250 kW erp). The authors noted that the groups were not mutually exclusive because some transmitters could be included in more than one group. The groups were chosen to examine the possibility of a strong dose-response effect or threshold rather than for independent tests of hypotheses.

To test the sites for an association of adult leukemia, skin melanoma, and bladder cancer with nearby residence, reported for the Sutton Coldfield site, the site was excluded from that analysis. For childhood leukemia and brain cancer, reported as not significantly in excess at that site, the site was included in the analysis.

Two of the sites were 4.3 km apart and two others were 15.4 km apart. The distances between all other site pairs were greater than 20 km. As in the Sutton Coldfield study, the area chosen around each site was a circle 10 km in radius, and each circle was divided into bands at radii 0.5, 1.0, 2.0, 3.0, 4.9, 6.3, 7.4, 8.3, 9.2, and 10 km. Also as before, comparisons were made between the results for the area within the arbitrarily chosen 2-km radius around each site and for the entire 10-km area. The larger population within all of the areas considered was about 3.39 million persons, based on the 1981 census.

Cancer registration data were derived from postcode residences of the subjects at diagnosis time for the periods 1974-1986 in England, 1974-1984 in Wales, and 1975-1986 in Scotland. Displayed in Table 1 of the paper were the results for "all leukemia", "skin melanoma", and "bladder cancer" (excluding the data from the Sutton Coldfield site), with "all leukemias" divided into the subcategories "all acute", "acute myeloid", "acute lymphatic", "chronic myeloid", and "chronic lymphatic". Those results were expressed in

terms of observed cases (O), expected cases (E), O/Es, and 95% confidence intervals (CIs) for the 2-km distances from the sites, and similarly for the 10-km distances.

The O/Es for the subjects within 2 km of each site ranged from 0.63 (for "chronic myeloid" to 1.20 for "chronic lymphatic"), but all of the CIs, including those for skin melanoma and bladder cancer, straddled 1.00 (non-significant). The corresponding results for those within 10 km of their sites were all slightly above 1.00 except for skin melanoma, for which the O/E was 0.90, and interestingly with a CI below 1.00 (0.85-0.94). The results for bladder cancer were O/E=1.09, CI=1.06-1.11, a statistically significant but hardly robust result. Similarly, the O/E for "all leukemias" was 1.03 and the CI was 1.00-1.07. Under "all leukemias", the subcategories "all acute" and "acute myeloma" had CIs of 1.00-1.11 and 1.00-1.13, respectively.

Shown in Table 2 of the paper were the O/Es and cumulative O/Es for the incidence of all cancers within each distance band for all of the transmitters combined, for the four defined transmitter groups, and for three specific sites. As in the similar table of the other paper, no CIs were given. The O/Es ranged from 0.87 for distances up to 1.0 km, with the largest O/E (1.15) for the distance band 2.0-3.0 km, and slightly above 1.00 for the remaining distance bands out to 10 km except an O/E of 0.96 for the 7.4-8.3 km band.

For Group 1, comprising the highest-power TV transmitters, the largest O/E was 1.29 for the distance band 2.0-3.0 km, with corresponding cumulative O/E of 1.17, encompassing 191 observed cases and 163.6 expected cases within 3 km of those sites. Most of the O/Es for the bands beyond 3 km were slightly larger than 1.00, with no apparent decrease with distance. Within 10 km of those sites, there were totals of 2,042 observed cases and 1,940.3 expected cases. Similar results were obtained for the other three groups and the three specific sites.

In the abstract, authors stated: "The magnitude and pattern of risk found in the Sutton Coldfield study did not appear to be replicated. The authors conclude that the results give no more than very weak support to the Sutton Coldfield findings." Overall, no credence is given to the positive findings of either study.

Thus, the epidemiologic and other human studies above that sought an association between cancer and RFEMF exposure do not provide scientifically credible evidence that chronic exposure to levels below the ANSI/IEEE (1992) exposure guidelines induces or promotes cancer. It should be noted, however, that cancer has been reported in some studies from exposure to magnetic fields at powerline frequencies (50 or 60 Hz), but that such findings were not confirmed in other studies. Pending the results of further studies, those positive and negative findings at powerline frequencies remain controversial. In this context, frequencies in the RFEMF range as defined herein (3 kHz to 300 GHz) are 50 to 5 billion times higher than 60 Hz. Thus, whatever the outcome of the powerline controversy, the findings thereof would not likely be applicable to whether RFEMF exposure induces or promotes cancer, because of the basic, highly-frequency-dependent differences in the dielectric properties and physical mechanisms of interaction of electromagnetic fields with biological entities.

In several of the epidemiologic studies discussed above, the occupations considered by the authors included those that may have involved exposure to frequencies in the RFEMF range as defined herein, as well as to frequencies below that range. In such studies based on occupational titles, little if any data or assumptions about the levels or durations of actual RFEMF exposure were given. Other studies of possible associations between cancer incidence and electrical occupations have been conducted, but largely of subjects presumably exposed to powerline fields, such as those from low-frequency (e.g. 60 Hz) power equipment of various kinds, and with emphasis on the role of the magnetic fields. Analyses of such studies are outside the scope of this report, but may be the subject of a subsequent report by the authors.

3.2 RFEMF AND INDIVIDUAL HUMAN CANCER CASES

Archimbaud et al. (1989) reported on a 46-year old patient with acute myelogenous leukemia (AML) who, for about 22 years, had been repairing microwave generators of powers up to 3 kW in professional microwave ovens. During that period, he had repaired five to six generators a day for six days a week, and had tested each repair by switching on the unprotected generator for one minute. Thus, the patient estimated that his daily microwave exposure had been five minutes. The authors remarked that the patient had not been exposed to any known carcinogenic chemical in his employment.

The clinical tests performed on the patient that yielded the diagnosis of secondary AML were described in some detail. Various treatments of the patient, including two courses of chemotherapy and a bone-marrow allograft, were ultimately unsuccessful.

The authors, citing the studies indicated, remarked that: "Although exposure to microwaves is known to induce genetic damage in animal species [Roberts and Michaelson (1983) and Szmigielski et al. (1982)] and in human lymphocytes *in vitro* [Stodolnik-Baranska (1967)], large epidemiologic studies of the potential role of microwaves in carcinogenesis have yielded negative results [Robinette et al. (1980)] However, populations studied were not intensely exposed to this type of radiation."

Jauchem (1990a) took issue with the remark of Archimbaud et al. (1989) about the suggested involvement of RFEMF-exposure in their findings, noting the absence of measurements of the RFEMF levels or of any other details regarding the patient's exposure conditions. Also questioned were the statements about the genetic activity of microwaves and interpretation by the authors of the references they cited. Jauchem (1990a) remarked: "Readers should exercise caution in making broad conclusions concerning the safety of exposure to microwaves on the basis of speculative and unsubstantiated reports."

Archimbaud (1990) responded to the comment of Jauchem (1990a) about the genetic activity of microwaves by citing the studies by Garaj-Vrhovac et al. (1990a), and Balcer-Kubiczek and Harrison (1989). [Garaj-Vrhovac et al. (1990a) had reported that cultures of V79 Chinese hamster cells exposed to 7.7-GHz RFEMF at 30 mW/cm^2 for 30 minutes exhibited inhibition of tritiated thymidine uptake and higher incidence of chromosome aberrations than control cultures. Balcer-Kubiczek and Harrison (1989) had reported that 2.45-GHz RFEMF, together with a known chemical carcinogen (TPA), promotes neoplastic transformation in mouse-embryo-fibroblast-cell cultures, whereas the RFEMF alone does not. The findings of both of these studies have been questioned by others. Jauchem (1991) also disputed other comments by Archimbaud (1990), including a statement that "microwave ovens used for cooking are known to leak during ordinary use".

Hocking and Garson (1990) also questioned the findings of Archimbaud et al. (1989) for reasons similar to those of Jauchem (1990a). They also added remarks about the absence of ocular effects in the patient and the negative findings of their cytogenetic study of 38 radiolinemen exposed at levels not exceeding the 1985 Australian standards (Garson et al., then in preparation, published in 1991).

3.3 SUMMARY OF EPIDEMIOLOGIC AND OTHER HUMAN RFEMF/CANCER STUDIES

Polson and Merritt (1985) found that the Lester and Moore (1982a) study of cancer incidence near Air Force bases (AFBs) was flawed because the authors had assembled a faulty database. When Polson and Merritt (1985) corrected the database, they found no association of cancer in populations living in the vicinity of "radar" AFBs. The Lester and Moore study (1982b) of cancer incidence from the radars at Wichita Mid-Continent Airport and McConnell AFB was also flawed for several reasons, but basically because their formula for assessing population exposure to RFEMF was not based on physical laws of RFEMF propagation.

The findings of Milham (1982) of leukemia from presumed occupational exposure to electric and magnetic fields were questioned by Liburdy (1982), based on citations to studies that do not support

those findings. The Milham (1982) findings are also questionable because of use of the "proportionate mortality ratio" (PMR) rather than a more appropriate statistic such as the "standardized mortality ratio" (SMR). Similar conclusions are applicable to the leukemia studies of Wright et al. (1982) and McDowall (1983). In none of those studies were any data presented on measured or estimated exposure levels or durations. Some excess risks for leukemia in electrical occupations were reported by Coleman et al. (1983), but there were no leukemia cases in the occupations "radio/radar mechanics" or "professional electronic engineers".

The findings of the more extensive Milham (1983) study of decedent data in the State of Washington are also questionable because of use of the PMR and because of possible personal bias of the author regarding the subjects in the various occupations.

The Milham (1985) study of deceased amateur radio operators reported a significant excess of leukemia deaths, but the numbers of cases were small (24 of 280 decedents versus 12.6 expected deaths), and as before, the use of the PMR is questionable. Also, Wangler et al. (1985) disagreed with the Milham (1985) findings, primarily because of the short time intervals spent by most amateur radio operators in actually transmitting, and the low average radiated powers during transmission. However, Coleman et al. (1985) disagreed with the Wangler et al. (1985) comments.

Milham (1988a, 1988b) used the SMR in the studies of decedent amateur radio operators, but little credence can be given to the findings because the numbers of deaths were small relative to the actual and expected totals.

The case-control studies of Pearce et al. (1985) and Pearce (1988) of leukemia patients yielded significant leukemia excesses for those in the occupations "radio/television repair" and "electrician," but the small numbers of cases render the findings questionable. A similar conclusion is applicable to Pearce et al. (1989) and Reif et al. (1989), the other studies by those authors. On the other hand, although the Thomas et al. (1987) study of cancer decedents yielded a statistical association of tumor incidence with their occupational histories, the authors did not regard RFEMF as the responsible agent because the subjects also may have been exposed to potential chemical carcinogens.

In the Hayes et al. (1990) case-control study of primarily military patients with testicular cancer, the authors reported higher risks for seminoma and "other" germinal cell carcinomas in various occupational categories. Included were patients with presumed "exposure to microwaves or other radio waves," but no excess risk was found for those in the "radar equipment" occupation. Unclear is why the authors regarded those two occupational categories as distinct. Moreover, an independent risk assessment by an industrial hygienist presented by the authors did not support their finding of higher risk.

In the Dolk et al. (1987a, b) studies of cancer incidence in the regions around high-power broadcast transmitters in Great Britain, the authors reported several positive findings of cancer incidence, but those findings were questionable for various reasons. Thus, no credence can be given to their positive or negative findings.

Archimbaud et al. (1989) reported an association of acute myelogenous leukemia (AML) in a patient who repaired high-power microwave generators and tested them unshielded. Jauchem (1990a) and Hocking and Garson (1990) criticized that finding, primarily because of the absence of measurements of the RFEMF levels or any other specifics about the patient's exposure conditions. On the other hand, the Garson et al. (1991) study of radiolinemen for possible chromosomal damage from chronic RFEMF exposure at levels within the ANSI (1982) and Australian exposure guidelines (AUS 1985) yielded negative findings.

Table 29 (A through Q) briefly display in chart form the findings, discussed above, of the various epidemiologic studies directed toward seeking a possible relation between RFEMF exposure of humans and cancer incidence. Also included is the Archimbaud et al. (1989) study of an AML patient discussed above.

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Robinette and Silverman (1977)	RFEMF-related higher mortality.	Occupational exposure to RFEMF of personnel who served in the U.S. Navy during the Korean war (levels and durations not estimated).	No significant differences were found between the high-exposure and low-exposure groups in deaths from all causes. The death rates from trauma were significantly higher in the high-exposure group, but were not RFEMF-related.	The high-exposure group was picked by occupational titles Electronics Technician, Fire Control Technician, Aircraft Electronics Technician. The titles of the low-exposure group were Radioman, Radarman, Aircraft Electrician's Mate.
Silverman (1979) [Review paper]	See Robinette and Silverman (1977).	See Robinette and Silverman (1977). Some in the high-exposure group were exposed at levels exceeding 10 mW/cm^2 at some times on some ships, but those in the low-exposure group were exposed at well below 1 mW/cm^2.	There were no significant RFEMF-related differences between the high-exposure and low-exposure groups in long-term-mortality or hospitalization patterns. No morbidity data were presented in Robinette and Silverman (1977) or in this review paper.	Other Naval personnel were added to those included in Robinette and Silverman (1977).
Robinette et al. (1980)	Same as Robinette and Silverman (1977). In addition, the numbers of admissions to Naval hospitals in various periods and admission rates for both groups were compared by ICD* diagnoses.	See Robinette and Silverman (1977).	Only 2 of the 18 comparisons between the high-exposure and low-exposure groups were significant: The low-exposure group had higher admission rates for mental disease, and for accidents, poisonings, and violence. The admission rates did not differ between groups for any disease class.	ICD = International Classification of Disease. Morton (1981) questioned the basis for selecting the high-exposure and low-exposure groups. Robinette (1981) noted the basis for their selection.
Lester and Moore (1982a)	Increased cancer incidence in areas surrounding U.S. Air Force bases (AFBs). The authors found 92 counties that had 1 or more AFBs, and matched each county with one of about the same population that had no AFBs.	RFEMF from the radars at AFBs. (Specific characteristics not given.)	The authors used a 1-tailed test for correlated proportions. They concluded that AFB counties had a significantly higher cancer mortality incidence for the period 1950-1969 than population-matched nonAFB counties, and they suggested that the higher incidence was due to RFEMF-exposure from the radars.	The authors indexed the data on cancer mortality in each AFB and nonAFB county in terms of severity relative to cancer mortality in the general U.S. population. Their findings (if valid) do not confirm that the increased cancer mortality is associated with RFEMF-exposure, but only that such mortality may be correlated with the presence of an operating AFB.

TABLE 29A: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Polson and Merritt (1985)	These authors carried out an independent reanalysis of the raw data provided by Dr. Lester on this study. The review of the raw data showed several inconsistencies, mostly in AFB location and counting, which were corrected.	The authors sought a possible geographic pattern of cancer incidence in Wichita, and whether specific RFEMF sources could be identified and related to any such pattern. Their analysis was based on morbidity data for first diagnosed cancer cases of Wichita residents for 1975, 1976, 1977.	Statistical treatment of the corrected data base showed that the incidences of cancer mortality in counties with AFBs for the period 1950-1969 did not differ significantly from the incidences in the nonAFB counties most closely matched in population.	In a response to Polson and Merritt (1985), Lester (1985) took issue with several aspects of the reanalysis and did not accept their finding of no significant differences in cancer-mortality incidence.
Lester and Moore (1982b)	The authors sought a possible geographic pattern of cancer incidence in Wichita, and whether specific RFEMF sources could be identified and related to any such pattern. Their analysis was based on morbidity data for first diagnosed cancer cases of Wichita residents for 1975, 1976, 1977.	The authors suggested that the radars at Wichita Mid-Continent Airport and McConnell AFB were the primary RFEMF sources, and they presented a formula relating incidence of cancer to terrain and exposure via line-of-sight transmissions to relatively high locations.	The authors selected 76 of the city's 94 census tracts, and assumed the incidence rates for the 76 census tracts to be the ratio of the number of cases in each tract to its population. They also obtained mortality data for all cancer deaths in those years, and analyzed incidence rates, age, economic status, male/female ratio, and race to obtain a correlation matrix for those 76 census tracts. Their finding was that cancer incidence in Wichita appears to be related to the probability of exposure to the RFEMF from the radars at the two airports.	This paper contains a number of flaws, among them the assumption that the population is exposed only to the RFEMF from radars at the two airports adjacent to the city, without providing measurements to support the assumption or any indication that the scan sectors of such radars were considered. Moreover, their exposure formula was not based on the physical laws of RFEMF propagation, most particularly the inverse-square-law of attenuation with distance. Thus, little if any credence can be given to their finding.
Milham (1982)	Leukemia in men from presumed occupational exposure to electric and magnetic fields in Washington State for the years 1970-1979. The author determined the number of deaths from each of 158 causes in each of 218 coded occupations.	No exposure data were given; assumption of field exposure was based on 11 of 218 occupational codes.	The PMRs for 7 of the 11 assumed field-exposure occupations were marked by the author as not statistically significant. For the remaining 4 occupational codes, (electricians, television and radio repairmen, power-station operators, and aluminum workers), 3 of the PMRs for "all leukemia" were marked significant; the exception was for the television and radio repairmen. Three of the PMRs for "acute leukemia" in those 4 occupations were also marked significant, but the nonsignificant exception was for power-station operators.	The author used the ratio of the number of deaths from each cause in each occupation to the expected number of deaths from that cause, multiplied by 100, called the "proportionate mortality ratio" (PMR). He surmised that the leukemia findings were associated primarily with exposures to large dc currents and high alternating electric and magnetic power fields. Liburdy (1982) disagreed with those findings, citing several references that do not support the leukemia association.

TABLE 29B: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Milham (1983)	The author gathered decedent data for the years 1974-1979 in Washington State, and analyzed the causes of death for 219 male and 51 female occupations. His report contained commentaries that described mortality patterns in each of the male and female occupation groupings.	The author suggested that some occupations may have involved exposure to electric and/or magnetic fields.	Some commentaries appear to be highly subjective and to reflect the personal biases of the author. On only one page of the report, 11 categories of workers presumed to be occupationally exposed to magnetic or electrical fields were grouped, and PMRs for two leukemia categories (acute leukemia, and all leukemia) were presented. Significantly high PMRs were reported for 5 of the 22 occupational-leukemia categories, but were probably significant because PMRs in some other categories were abnormally low. Thus, the association found between leukemia and exposure to electric and/or magnetic fields is questionable.	Described were the occupation codes used in preparing death certificates, the occupation groupings for women, and the occupation indexes for men and women. The author used the "proportionate mortality ratio" (PMR), making their values interdependent. Use of the "standardized mortality ratio" (SMR) would have been more appropriate; it indicates the percentage of deaths for each cause to the expected number of deaths from that cause, independent of any other SMR.
Wright et al. (1982)	Leukemia in men from presumed occupational exposure to electric and magnetic fields during 1972 to 1979, as determined from the Cancer Surveillance Program in Los Angeles County.	No exposure data were given.	A trend toward a higher risk of leukemia from occupational exposure to electric and magnetic fields was reported, with the greatest risk for acute myelogenous leukemia (AML).	The authors used proportional incidence ratios (PIRs) for the data in the Los Angeles County Cancer Surveillance Program. The authors remarked that the subjects may also have been exposed to metal fumes, solvents, fluxes, chlorinated biphenyls, synthetic waxes, epoxy resins, and chlorinated naphthalenes, a point that render the findings highly questionable.
McDowall (1983)	PMRs for occupational mortality in England and Wales during 1970 to 1972 in males 15-72 years of age. A case-control study of the deaths of 537 males from AML in the year 1973, with 1074 male deaths from all causes except leukemia as controls.	No exposure data were given.	The PMR study showed that the mortality distribution for all leukemias did not differ significantly from expected, but the PMRs for AML were significantly higher than 100 for self-described electrical engineers, telegraph radio operators, professional electrical engineers, and professional electronic engineers. The results for the case-control study showed significantly elevated relative risks (RRs) for the electrical occupations, with the highest RR for telecommunications engineers.	It is noteworthy that the PMRs for occupations that may have involved exposure to RFEMF (as defined herein) ranged from a low of 61 for radio and radar mechanics to a high of 249 for telegraph radio operators. However, as with other studies in which the PMR statistic was used, both the positive and negative findings of this study are open to question.

TABLE 29C: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Coleman et al. (1983)	Leukemia incidence in males of ages 15-74 years in 10 electrical occupations in South East England during 1961-1979. The authors remarked that the South Thames Cancer Registry for 1961-1979 listed about 30,000 tumors per year in a population of 6.5 million; proportional registration ratios (PRRs) for leukemia were calculated in the males therein.	No exposure data were presented.	Found was a 17% excess of all leukemias in all electrical occupations (PRR=117, $p<0.05$). However, although 8 of the 10 occupations showed excesses, there was an all leukemia deficit (PRR=22, $p=0.07$) for "radio/radar mechanics". The PRRs for all of the electrical occupations also showed no overall excess of chronic myeloid leukemia, but a 46% excess of acute lymphoid leukemia, a 29% excess of chronic lymphoid leukemia, and a 23% excess of acute myeloid leukemia (AML). The excesses of AML were in 7 of the 10 occupations, but not all the excesses were significant, and there were no cases in the occupations "radio/radar mechanics" or "professional electronic engineers".	The authors' null hypothesis was that the proportion of leukemic men in each 5-year-age group in each electrical occupation would be the same as the proportion of the men with leukemia in all occupations. The results show no evidence of excess leukemia associated with those occupations that may involve exposure to RFEMF.
Milham (1985)	Mortality data on the male members of the American Radio Relay League (operators of amateur radios) listed as decedents in QST, the monthly magazine of the League.	No specific exposure data were presented. The author noted that some ARRL members also may have been in occupations involving RFEMF exposure.	The PMR was 281 for acute, chronic, and unspecified myelogenous leukemia; the PMR for monocytotic leukemia was only 77 (well below 100), and there were no cases of lymphatic leukemia. Thus, the PMR for all leukemias was 191 (24 deaths versus 12.6 deaths expected, $p<0.01$).	Death certificates for 1971-1983 were obtained for 280 decedents in the State of Washington, and information on the age, date, and cause of death was obtained for 1411 decedents in California. PMRs were calculated for specific and all leukemias. As noted previously, the use of the PMR statistic is questionable.
Wangler et al. (1985) Coleman (1985)	For several reasons, Wangler et al. (1985) took issue with the Milham (1985) finding of a higher risk of leukemia in amateur radio operators. Coleman (1985) did not agree with most of the comments by Wangler et al. (1985).	See Milham (1985).	See Milham (1985).	Wangler et al. (1985) cited a 1980 survey in Canada and the U.S. that showed that typical amateurs spent 6.1 hours per week on that activity, much of which was highly variable and most of which was listening, with the remainder involving intermittent transmissions at low average radiated powers. They also questioned the statistical treatment used.

TABLE 29D: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Milham (1988a)	Mortality in amateur radio operators due to RFEMF-exposure from their transmitters. The author obtained the names of 67,829 males licensed in California and the State of Washington, and searched them for deaths during 1979-1984, which yielded 2,485 decedents taken to have had 232,499 person-years at risk.	Specific frequencies were not indicated, but presumably were in the FCC-approved amateur bands.	Unlike in his previous studies, Milham (1988a) used the SMR. For "all malignant neoplasms," the SMR was 89 with an 82-95 CI, a significantly lower death rate than for the general population. The SMR for the subcategory "other lymphatic tissue" of malignant neoplasms was 162 with a CI of 117-218, a significant excess. Also examined were 9 leukemia subdivisions or sub-subdivisions. Of 36 deaths, 15 were for "acute myeloid" leukemia versus 8.5 of the expected 29. The SMR was 176 with a CI of 103-285, also a significant excess.	An SMR that exceeds 100 with a 95% confidence interval (CI) entirely above 100 is deemed a significant increase; an SMR less than 100 with a CI below 100 is a significant decrease. Little credence can be given to the significant excesses found, in view of the small numbers of deaths relative to the actual and expected totals.
Milham (1988b)	Mortality in amateur radio operators in the 5 FCC-license classes: novice, technician, general, advanced, and extra.	Specific frequencies were not indicated, but presumably were in the FCC-approved amateur bands.	The numbers of deaths and SMRs in each class from all causes, all malignant neoplasms, and various specific types of malignancies were tabulated. The SMRs for all death causes were significantly below 100. For deaths from all malignant neoplasms, the SMRs for all 5 license classes were below 100, but the collective SMR for the malignant-neoplasms category was 89. The SMRs for lymphatic and hematopoietic neoplasms exceeded 100, but the only significant excess was for the technician-license class. Also, except for the novice class, the SMRs for multiple myeloma and other lymphomas exceeded 100, but the excess was significant only for the general-license class.	No confidence limits were shown, but the significant SMRs were so marked. In both the technician-license and general-licence classes, for which significant excesses were reported, the numbers of deaths were small: 18 of 409 deaths in the former class and 15 of 862 deaths in the latter class.

TABLE 29E: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Pearce et al. (1985) Pearce (1988)	From a case-control study of 546 male leukemia patients for association with various agricultural occupations in New Zealand (NZ), Pearce et al. (1985) sought an association with several specific occupations that may have involved exposure to electric and/or magnetic fields.	The patient data for the study were drawn from the NZ Cancer Registry. Tabulated from the case-control study were leukemia odds ratios (ORs) and 95% confidence limits (CIs) for patients in 10 occupational classes, a total of 18 of the 546 cases. The controls were 43 of the 2,184 controls in thelarger study. No data were given on actual exposures to electric or magnetic fields.	Significant leukemia excesses were shown for electronic equipment assemblers, and in the radio/television repair category. The authors suggested that the excesses were due to exposure to nonionizing radiation or to metal fumes and other substances used in electrical components or their assembly. Several corrections were given in Pearce (1988), primarily that the excesses should have been ascribed to the radio/television repair and electricians occupations, and that there were no cases in the electronic equipment assemblers category.	The findings are questionable from a statistical viewpoint, primarily because of the small numbers of cases.
Pearce et al. (1989)	A case-control study of 488 patients in recorded electrical occupations out of 19,904 male patients with all types of cancer.	No data were given on actual exposures to electric or magnetic fields.	Of the 22 sites tabulated, 11 had ORs less than 1 and the other 11 had ORs in the range 1.0-1.62. However, all of the latter sites had CIs that spanned 1.0 (nonsignificant), except for leukemia, which had 21 cases versus 13 expected, OR=1.62, CI 1.04-2.52. In the specific occupations, the largest OR was for radio/television repair, in consonance with the previous finding for that occupation, but the OR for electricians was not significant, in contrast to the previous finding. For the five leukemia subtypes studied, the only significant excess was for chronic-lymphatic rather than acute-myeloid leukemia reported in other studies. Also noteworthy was no excess of brain cancer.	The controls for the patients with cancer at any specific site or cancer type consisted of those with cancer at other sites. The findings of this study are questionable for the same reasons as for the previous studies by these authors. In this paper, moreover, they did note that many comparisons involved small numbers.
Reif et al. (1989)	Incidences of brain cancer by major occupational category, and in 3 occupational subgroups.	The patient data were also drawn from the New Zealand Cancer Registry.	Highest incidence of brain cancer was for "agricultural workers, forestry workers, and fishermen," with an overall OR of 1.38 and a CI of 1.08-1.77. Only within the third-highest-incidence subgroup, "production workers," were there 8 of 185 cases classified as "electrical workers"; with an OR of 0.78, and a CI of 0.39-1.59.	As with the other studies of these authors, discussed above, little if any credence can be given to either the positive or negative findings.

TABLE 29F: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Thomas et al. (1987)	Brain tumor mortality from occupational exposure of men who had died at age 30 or older from brain tumors or other tumors of the central nervous systems.	Job categories or job codes were assigned to occupational histories to assess presumed exposure to: RFEMF, lead, and soldering fumes.	Data were obtained about men who had died from tumors during a 3-year period. One control for each case was selected from men matched in age and year of death and area of residence, but who had died from other than brain tumors. Of 435 cases, 300 had astrocytic tumors, 90 had other types of tumor cell, and 45 had unknown types of tumor cell. Highest relative risk (RR) was for the combined categories of engineers, teachers, technicians, repairers, and assemblers. Significant RRs were also reported for those exposed to soldering fumes.	The authors remarked that RFEMF was not the responsible agent because those exposed to RFEMF also may have been exposed to lead, solder fluxes, solvents, and other chemicals.
Garland et al. (1987)	Noting that Hodgkin's disease might result from previous illnesses, the authors sought possible increased incidence of the disease in active-duty naval personnel during 1974-1979 from occupational exposure, relative to the general population..	Compared were the rates of first hospitalizations for Hodgkin's disease in the naval personnel with general-population data derived from NCI's SEER database and with the total Navy rates.	The overall results were an age-adjusted incidence rate of 2.9 cases per 100,000 person-years for the naval personnel and 3.7 cases per 100,000 person-years for the general population. By use of the standardized incidence rate (SIR) and 95% confidence limit based on the Poisson distribution, the difference was found to be nonsignificant.	Although not noted by the authors, the occupations tabulated that may have involved exposure to RFEMF included "radioman", "electronics technician", "airman", "fire control technician", and perhaps others, which showed no significant excess. The only statistically significant excess was for the occupational group "machinist's mate", a finding ascribed to exposure to various chemical and physical agents in close quarters.
Garland et al. (1988a)	A similar study of non-Hodgkin's lymphomas.	See above.	No excesses of non-Hodgkin's lymphomas were found in any of the occupations tabulated.	In actuality, the incidence of non-Hodgkin's lymphomas for the naval personnel was significantly lower than for the general population.
Garland et al. (1990)	A study of leukemia incidence in white-male, active-duty naval personnel during 1974-1983.	The data were derived from the computerized "Inpatient Follow-up Data System" maintained at the Naval Health Research Center in San Diego, and were compared with the NCI SEER database and with the total naval population.	Tabulated were the numbers of men by each diagnostic type of leukemia, totaling 102 verified cases out of 123 first hospitalizations for leukemia. In the acute-leukemia subcategory, the larger incidences were for lymphoid (19 cases) and myeloid (18 cases). There were also 19 cases of chronic myeloid leukemia and 12 cases of unspecified myeloid leukemia; all other leukemia subcategories had 9 or fewer cases each. The only significant SIR relative to the SEER or the total naval population was for "electrician's mate", which had SIRs and lower 95% confidence-interval limits exceeding 1.0. However, the tasks of those men primarily involved electrical equipment [ELF].	The authors noted that like other reported occupational studies of the possible association of [60-Hz] electric or magnetic fields and leukemia, this study had no direct measure of exposure. As in Garland et al. (1987), some of the tabulated occupations that may have involved RFEMF exposure showed no significant excess leukemia incidence.

TABLE 29G: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Garson et al. (1991)	Sought was chromosomal damage in blood samples from about 250 randomly-selected radiolinemen employed at Telecom Australia to erect and maintain RFEMF transmissin towers for radio and TV broadcasting, telecommunications, and satellite uplinks, and in blood samples from 40 unexposed clerical persons matched by age and Australian State.	The exposure frequencies ranged from 400 kHz to 20 GHz. Before 1985, the exposure levels were within the ANSI/IEEE (1992) guideline. After 1985, they were within the occupational limits specified in the 1985 Australian standard [AUS, (1985)] which also has limits to avoid shock and burn.	Dosimetric measurements of the induced current flow in the legs of radiolinemen at sites operating at up to 100 MHz yielded values lower than the specified 100-mA limit for each leg. Duplicate blood samples drawn from each exposed and control subject were cultured, and 100 metaphases were counted and assayed for chromatid gaps, chromatid breaks, chromosome gaps, chromosome breaks, and "other" (more complex) abnormalities. Results were tabulated in terms of rates per 100 cells for each abnormality, the ratios of the rates for the two groups, and the 95% confidence intervals thereof. Most of the ratios of the exposed-to-control rates slightly exceeded 1.0, but all of the confidence intervals spanned 1.0, thus indicating that the differences between the exposed and control subjects were not significant (p>0.05).	The authors remarked that smoking, recent infections, and X-rays were possible confounding factors, and they therefore made adjustments for each such factor but not involving matched pairs (because of missing data on some subjects). They found that none of those factors materially altered the negative findings. They also indicated that one of the control subjects exhibited at least 30 chromatid breaks, 14 chromosome breaks, and 3 other aberrations, totaling at least 47 aberrations. The data for that control subject were excluded as outliers, but were included in the table without the 95% confidence intervals.
Garland et al. (1988b) [Brief report]	Testicular cancer in Naval enlisted active-duty white personnel during 1974-1979.	The histories and demographic data were derived from a computerized database having more than two million person-years at the Naval Health Research Center (NHRC) in San Diego, CA. Hospitalizations for testicular cancer for that period, numbering 143 cases, were identified in a medical history file also maintained at the NHRC.	The 143 cases were tabulated by age group. The largest incidence of enlisted persons was in the age group 20-24 years: 67 cases comprising 964,189 person-years at risk and an average annual incidence rate of 6.9 cases per 100,000 person-years. However, the average annual incidence rate for that age group derived from NCI's SEER database for 1973-1977 was 9.3 cases per 100,000 person-years. The next largest incidence was in the age group 25-34 years: 48 cases comprising 597,022 person-years at risk and an average annual incidence rate of 8.0 compared to 9.3 cases per 100,000 person-years in the SEER database. For the 143 cases overall, indirectly age-adjusted by reference to the SEER database, the mean annual incidence rates were respectively 3.7 and 3.9 cases per 100,000 person-years. Thus, no significant differences were found in age-adjusted or age-specific incidences of testicular cancer in those personnel.	Although 3 of the 110 occupations studied had elevated SIRs relative to SEER, those occupations involved daily exposure to a variety of chemical agents, many of which are carcinogenic, but would not have involved much if any exposure to RFEMF. Thus, neither the negative nor the positive findings of this study provide any data about a possible link between RFEMF exposure and testicular cancer.

TABLE 29H: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Hayes et al. (1990)	A case-control study of 271 patients in 3 medical centers who were newly diagnosed as having testicular cancer, to determine the occupational risk for that type of cancer relative to other types. The controls were 259 patients diagnosed at the same centers with cancer in other than the genital tract.	In patient interviews about occupational activities, they were asked about exposure to or contact with various materials and equipments as sources of ionizing and/or nonionizing radiation, and to pesticides or polycyclic aromatic hydrocarbons.	A general finding was a higher risk for seminoma (OR=2.8) in all professional subcategories. No increased risk for seminoma was associated with the blue-collar occupations, and the risk was significantly lower for agricultural, forestry-and-fishery, and construction workers. Noteworthy were the results for "exposure to microwaves or other radio waves": OR=3.1 (1.4-6.9 CI) for all testicular tumors. Also, when only the subjects treated in the 2 military hospitals were considered, the OR for all testicular tumors was 3.5 with a CI of 1.3-10.2. Similar results were shown for seminomas and "other" germinal-cell carcinomas. However, for the category "radar equipment," the OR for all testicular tumors was only 1.1 with a CI of 0.7-1.9 (nonsignificant). Not clear is why the authors did not regard the two categories "exposure to microwaves or other radio waves" and "radar equipment" as one.	Two of the 3 medical centers were military hospitals, and many of the patients were military personnel on active duty. Results were tabulated by occupation in terms of the odds ratios (ORs), 95% confidence intervals (CIs), and numbers of controls for all testicular cancers combined, and by subdividing the cases into seminomas and "other" germinal-cell carcinomas. The authors also presented an independent assessment by an industrial hygienist that did not support the higher risk for the category "exposure to microwaves or other radio waves". Thus, the results do not show an increase of risk of testicular cancer relative to the other types of cancer from occupational exposure to RFEMF, and do not provide either positive or negative evidence that the other types of cancer suffered was due to chronic RFEMF exposure.
Davis and Mostofi (1993) [Brief report]	Six testicular cancer cases (all white males) reported between 1979 and 1991 among a cohort of 340 officers in two police departments within unidentified contiguous counties in the north-central U.S.	The occupational practice of the cases was resting the radar gun while in the 'on' position, directly in the lap either close to, pointing at, or directly adjacent to the testicles. Three of the six cases had embryonal cell carcinomas, one was diagnosed as having a mixed cell carcinoma with an embryonal cell metastasis, and the other two had seminomas.	Based on interviews, the authors assumed that the sizes of the police departments were stable at 340 men. From use of the 1981 SEER registry rates for white men of ages 20-49 years and obscure reasoning, they calculated that the number of expected cases of testicular cancer in a cohort of men who were followed from 1963 through 1991 would be 0.87 (O/E=6.9, $p<0.001$, Poisson distribution), but they did not provide any numerical data about the calculation.	The authors, citing Rothman (1990), noted that any time-space aggregate of disease can be defined so as to produce an elevated cancer rate in any cluster, and they offered a brief but unclear discussion about how they avoided this pitfall. They also remarked that testicular cancer had been noted among microwave radar operators, but the reference they cited [Mostofi and Davis (1989)] could not be found. The findings in this report provide no credible evidence that the incidence of testicular cancer in the 6 police officers was associated with their exposure to the RFEMF from the traffic radar guns they had used.

TABLE 29I: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Fisher (1993)	Measured were the X-Band and K-Band RFEMF levels emitted by 30 models of police traffic radars from 6 manufacturers. Included were hand-held devices and those with antennas in fixed configurations. The total number of antennas evaluated was 5000 (1075 for X-Band and 3925 for K-Band). The data were collected in 1982-1991. About 95% of the units had fixed-mounted antennas, and the remaining 5% were hand-held devices.	Used mostly was a Narda Model 8621B isotropic probe with its its sensors at the center of a 5-cm-radius polystyrene sphere. Each antenna's output was measured at its aperture with the spherical surface of the probe in contact with the aperture, and hence in the near field. Such measurements were useful for comparisons among the antennas and for relating them to far-field values.	The major results were tabulated as average output power densities and standard deviations for the individual manufacturers (coded A1-A11). The values ranged from 0.28 ± 0.03 mW/cm^2 for A5 to 3.36 ± 0.56 mW/cm^2 for A4 (both X-Band radars with fixed-mounted antennas). For A9, the only hand-held category listed (142 devices for K-Band), the average output power density was 0.44 ± 0.07 mW/cm^2.	The author cited the safety standards for the relevant frequency bands during the years covered [ANSI (1982) and ANSI/IEEE (1992)] and stated: "We are able to conclude with a high degree of certainty that there is no evidence to support the allegation that police traffic radar operators are at risk due to prolonged exposure to microwave emissions from their radar units."
Balzano et al. (1995)	Equivalent power density measurements were done at the antenna aperture of a Kustom Signals Falcon 24-GHz hand-held traffic radar, and of penetration depth and energy deposition in wetted chamois skin, a material of RF-absorption properties similar to those of human skin at that frequency.	The authors remarked that previous measurements by others with E-field probes of relatively large sizes in the plane of an aperture were likely in error. They used an EIT Model 979 E-Field Probe with sensors only 2.5 mm long, about 1/6 of the wavelength at 24.15 GHz (12.5 mm), and a NARDA/BRH Model 14 Implantable Probe with sensors 1 mm long, less than one-twelfth of that wavelength.	As expected in the near-field zone, a spatial plot of E^2 within the horn face (r=0) was a very complex pattern. A plot at r=5 cm was far less complex, but with a dip in the center of the pattern. At 9.5 cm, the pattern was much smoother and without the central dip. A plot of E^2 versus distance along the axis indicated that the far-field zone starts at about r=15 cm. Measurements of forward, reflected, and radiated powers with the radar horn radiating into free space and with the horn aperture held or pressed against various parts of the human body showed that RF energy at 24 GHz is readily absorbed within the body, with only a small fraction reflected back. For one layer of 0.4-mm-thick wetted chamois skin over the horn face, E^2 at r=2 cm showed substantial attenuation relative to that in the absence of the chamois, and even more so with two layers.	The authors found that for the 12-mW radar unit, the highest measured power density incident on the skin when the antenna is in close proximity was less than 0.5 mW/cm^2; and that more than 95% of the energy is absorbed in the first millimeter of depth when the antenna is placed in contact with tissue. The authors also remarked that the penetration depths for infrared (IR) wavelengths are comparable to those measured at 24 GHz in this study, and found it interesting that the maximum allowable exposure from IR lasers is 100 mW/cm^2 (ANSI, 1993). In overall conclusion based on the analyses of the foregoing studies, there is no scientifically credible evidence to support an association of testicular cancer with RFEMF exposure.

TABLE 29J: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Schlehofer et al. (1990)	A case-control study of the occupational risk factors for brain tumors for years 1987-1988 in a specific region of the Federal Republic of Germany then containing 1.3 million inhabitants.	Ninety-nine males and 127 females with brain tumors were selected from the two local neurosurgical clinics. Of those, 51% had glial tumors, 56% meningeal tumors, and 13% "other" tumors. The controls, numbering 418, were randomly selected from the local residental registers, and frequency-matched by age and gender. Data were collected from both groups on residential history, occupation, smoking, previous diseases, drug consumption, and nutritional habits.	A major finding was no elevated brain-tumor risk for smoking. More pertinent were the data for occupational exposure in 16 categories, one of which was "electricians" (for 5 or more years). For both genders, there were 13 cases of brain tumor (6%) and 14 controls (3%). The relative risk (RR) was 1.87 with a 95% confidence interval (CI) of 0.9-4.1 (nonsignificant). The 13 cases consisted of 6 females and 2 males with 5-20 years, and 2 females and 3 males with more than 20 years, as electricians. The only significant finding was for the 6 females with 5-20 years of experience: RR=6.22 (relative to 2 controls) and a CI of 1.2-31.7. For the 2 females with more than 20 years, RR=4.56 and CI=0.4-31.9 (nonsignificant). The results for all 8 female cases were shown as RR=5.2 and CI=1.4-20.1 (significant), but the authors recognized that the numbers of cases were too small to ascribe any credence to this single positive finding.	As with a number of epidemiologic studies that coveried a broad range of possible agents, any findings (whether positive or negative) relative to RFEMF exposure provided little if any evidence for an association with cancer incidence.
Maskarinec et al. (1994)	The authors analyzed a cluster of leukemic children reported by a pediatric oncologist at the Waianae coast on Oahu, Hawaii. They investigated whether the incidences, derived from the Hawaii Tumor Registry during the period 1979-1990, could be ascribed to the presence of two low-frequency radio towers (frequencies not stated) in the region.	The cases studied were 12 children under 15 years old who had lived in three local census tracts and who were diagnosed with acute leukemia: one each during 1979, 1980, 1985, 1986, and 1990; two each during 1982 and 1984; and three in 1983. A case was defined as a child less than 15 years of age diagnosed with acute leukemia and who had lived in the region before diagnosis. The controls were 4 gender- and age-matched children who had resided in the town of Waianae at the time of diagnosis.	All case addresses were located, and their distances from the radio towers were estimated to within 0.2 mile. Matched odds ratios were calculated, using the SAS (1989) software program. The standardized incidence ratio (SIR) was 2.09 CI) of 1.08-3.65 (significant). The SIR and CI for acufor all 12 cases with a 95% confidence interval (te lymphocytic leukemia were 1.58 and 0.63-3.26 (nonsignificant), and were 3.73 and 1.20-8.71 (significant) for nonlymphocytic leukemia. The authors remarked that the statistical power of the findings was low because of the small number of cases, and they specifically stated: "It appears that closeness to the low-frequency radio towers has a weak association with leukemia, even though it was not statistically significant."	The authors' statement quoted has no scientific validity. They also noted that the EPA had measured the electric fields and magnetic fields around Lualualei Naval Station in 1990 and found that the levels did not exceed existing guidelines, citing EPA (1992). For both reasons above, no credence can be given to the findings about an association of leukemia with RFEMF exposure from the two towers.

TABLE 29K: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Cantor et al. (1995)	The authors sought whether a relationship exists between the incidence of female breast cancer and occupational exposure to various substances, ionizing radiation, and RFEMF.	More than 2.5 million mortality records from 24 states for years 1984-1989 were coded for occupation. Breast cancer was the underlying cause of death for 59,515 female decedents, of which 5,970 (10%) were black women. After exclusions, the totals were 33,509 cases and 117,794 matched controls of both races.	Tabulated were 31 potential workplace-exposure agents and odds ratios (ORs) for "probability of exposure" on a scale 0-4 and "level of exposure" on a scale 0-3, with 2 of the 31 categories dealing with RFEMF. The largest ORs were for solder, with 95% CIs above 1.0. Those for most other agents were below 1.2 with 95% CIs spanning 1.0 (nonsignificant). Regarding RFEMF exposure, a few ORs for women of both races were barely significant at exposure levels 1 and 3, but were nonsignificant at level 2, results correctly discounted by the authors.	The authors indicated the many limitations of their study, not the least of which was the inability to control for most recognized breast-cancer risk factors. They also stated that they had found no association of breast cancer with either ionizing or nonionizing radiation. This study yielded no positive or negative evidence of a relationship between exposure to RFEMF and the incidence of female breast cancer.
Tynes et al. (1992)	The authors sought the risk of cancer in a cohort of 37,945 electrical workers in Norway, 20-70 years of age, based on job descriptions derived from the 1960 census.	The authors linked 12 occupations to a registry of cancer mortality and morbidity, using the workers' personal identification numbers. Two of those occupations were "radio/telegraph operators" and "radio/television repairmen", respectively comprising 1,641 (4.3%) and 1,376 (3.6%) persons.	Calculated were the expected number of cancer cases in terms of 5-year, age-specific incidence rates for each year from 1961 through 1985, and the standardized incidence ratios (SIRs) and 95% confidence intervals (CIs). Tabulated were the numbers of observed cases of 25 different cancer sites, the corresponding expected numbers of cases, and the SIRs and CIs. The results for all cancers in all occupations (3,806 observed cases or about 10% of the entire cohort) were an SIR of 1.06 and a CI of 1.03-1.09. Shown separately were the results for leukemia (107 cases total), as "Acute" (lymphocytic, myeloid, other), "Chronic" (lymphocytic, myeloid), and "Other", and for brain tumors. All of the CIs spanned 1.00 except for myeloid in a small subcohort (74 cases) of persons still economically active in 1970. There were a total of 9 cases in the two RFEMF-related groups, from which the authors concluded that there was a significant excess risk of leukemia among workers in occupations with potential for exposure to RFEMF. The SIRs and CIs for brain tumors in the two RFEMF-related groups were nonsignificant.	The authors remarked that the term "electrical worker" is too vague to be a good marker for exposure to RFEMF, and that the results should be interpreted with caution in the absence of field measurements in the selected jobs. They noted that potential exposure of the workers to solvents, polychlorinated biphenyls, and soldering fumes may explain the excess risks found.. For the reasons indicated and the small percentages of cases relative to the entire cohort analyzed, little if any credence can be given to the findings of this study.

TABLE 29L: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Tynes et al. (1996)	Studied was breast-cancer incidence in Norwegian female radio/telegraph operators potentially exposed to light at night, RFEMF in the range 405 kHz to 25 MHz, and ELF (50 Hz) while on duty at sea. Hypothesized was that exposure may cause breast cancer by decreasing the production of melatonin, which would enhance the susceptibility of subjects to cancer associated with sex hormones.	The subjects were 2,619 women certified as radio and telegraph operators between 1920 and 1980, of which 98% had worked on Norwegiam merchant ships. The cohort was followed from 1961 through 1991. They were linked to a Norwegian Cancer Registry (1959 to derive the incidence of cancer in females. Derived also was a cohort including females in job titles other than radio and telegraph operators at sea. Spot RFEMF measurements were made aboard 3 ships.	Job histories were collected for the cases and controls. Shift work was categorized as 0, 1, 2, or 3 to indicate the extent of their presence in the radio room during the day and at night, the latter reflecting possible exposure to artificial light. In the analysis, the statistical unit was the number of years each employee was followed up. Each work year of each member was tallied by that person's age and calendar year, and the person-years of all members were summed. The SIR of cancer cases was calculated relative to the number of females in the national population. Follow-up yielded 72,105 person-years, during which 140 new cancer cases were observed and tabulated. The highest incidence was for breast cancer (50 women), with a SIR of 1.5 and a CI of 1.1-2.0. Next in decreasing order were cervical cancer (14 women, SIR: 1.0; CI: 0.6-1.7) and uterine cancer (12 women, SIR: 1.9; CI: 1.0-3.2). The other 64 cases were for other types of cancer, each with less than 10 women and nonsignificant SIRs.	It is difficult to understand why the authors regarded their results as indicating a significant relationship between breast-cancer incidence and occupational exposure to RFEMF and/or artificial light during night work in the radio room, based on a vague speculative tie between such exposure and inhibition of melatonin production, said to be effective in reducing the growth of melanoma. In addition, the authors recognized the limitations of their dosimetry, noting that no historical exposure data were available. Measurements at the operator's desks indicated that the RFEMF levels for all frequencies were lower than the occupational guidelines recommended by The International Radiation Protection Association.
Hocking et al. (1996)	The authors investigated whether people that lived in the vicinity of a triangle of 3 TV towers in northern Sydney, Australia, showed a higher incidence of cancer and mortality due to the RFEMF broadcasted by the towers than did the general public. The frequencies were in the range from 63 MHz to 215 MHz.	Studied were 135,000 people within the "inner area", defined as within a radius of roughly 4 km from the geographic center of the tower triangle. They were compared with 450,000 people in the six nearby municipalities, called the "outer area", based on assuming a decrease of RFEMF intensity by the inverse-square law.	The calculated intensity results were displayed as a graph of power density on a logarithmic scale versus distance from the tower center on a linear scale. The authors noted that the values were far lower than those in the 1990 Australian exposure standard, and that some measurements by the Commonwealth Department of Communications near one of the towers yielded values fivefold lower than calculated. The data for leukemia and brain tumor (the latter especially in children) were tabulated extensively by age groups, sex, years, and numbers of cases. The primary findings were that the incidence and mortality of brain cancer was not increased by proximity of the residences of the cases to the TV towers, but that the incidence and mortality of childhood leukemia was significantly higher in the inner area (and not in the outer area), presumed by the authors to be due to the RFEMF from the TV towers.	The authors mentioned various possible biases and confounding factors that may have affected the findings. However, even if the increase in childhood leukemia in the inner area were accepted, there is no clear evidence that the increase was related to the RFEMF from the towers. Also, the calculated power densities are questionable because those values differed widely from the measured values, a point noted by the authors. Thus, no credence can be given to RFEMF as the basis for any positive findings of this study.

TABLE 29M: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Szmigielski (1996)	Examined for cancer morbidity was the entire population of personnel with military careers in Poland during 1971-1985. The mean yearly count was about 128,000.	Each year, about 3,700 persons were selected on the basis of their service records and documented at service posts as having been exposed to radio frequencies (RF) and microwaves (MW). The author noted that at most of the posts, the fields (mostly pulse-modulated 150-3,500 MHz) did not exceed 2 W/m^2, (0.2 mW/cm^2). At the other posts, the fields were 2-6 W/m^2 (0.2-0.6 mW/cm^2), with frequent short-lasting exposures that exceeded 6 W/m^2.	The personnel in active service were divided into 10-year age groups: 20-29, 30-39, 40-49, and 50-59 years, and data on morbidity (first malignancy diagnosis) were derived for each age group and for the whole population. The neoplasms were classified in terms of 12 occurrence sites, and the numbers of exposed (observed) and nonexposed (expected) incidences per 100,000 each year were averaged over the 15-year period. The latter was taken as the incidence for the total population, including those exposed to RF/MW. Tabulated were observed-to-expected ratios (OERs) and 95% CIs of cancer incidence at the 12 sites. The OERs for the following sites were labeled as significant: esophageal and stomach, colorectal, nervous system (including brain tumors), hematopoietic system and lymphatic organs, and skin (including melanomas). All with malignancies (per 100,000) totaled 119.12 persons versus 57.60 expected, for an OER of 2.07 and a CI of 1.12-3.58 ($P<0.05$). Specific results were tabulated in more detail.	The author stated that a reasonable explanation could not be offered for a threefold increase of stomach and colorectal adenocarcinoma or any certainty about causal links with RF/MW exposures, noting that no differences could be found in life-styles or possible exposure to other carcinogenic substances. Not clear was how the numbers of unexposed persons who comprised the expected numbers used in calculating the OERs were derived. Not presented were the numbers of cases and controls each year and the specific kinds of duties performed by those at RF/MW posts versus those for non-RF/MW personnel at the same posts, to provide a better basis for assessing the findings.
Grayson (1996)	The author did a nested case-control study of brain-tumor risk from presumed exposure to non-ionizing radiation [ELF and RFEMF] and ionizing radiation of 230 men diagnosed for primary malignant brain tumor, taken from 880,000 Air-Force persons who had served at least one year during 1979-1989. The exposures to RFEMF were based on a registry of all incidents of Air-Force personnel possibly exposed at levels exceeding 10 mW/cm^2, and were used to categorize job titles for exposure severity over time.	Selected for each case were 4 controls matched for year of birth and race, and who were part of the total cohort when that case was diagnosed. Not eligible as controls were those who had been diagnosed with leukemia, breast cancer, or malignant melanoma.	Tabulated were the number of enlisted personnal by rank ("airmen", "non-commissioned officers", and "senior non-commissioned officers") and commissioned officers ("company-grade officers", "field-grade officers", and "general officers"), for a total of 61 cases and 138 controls. The age-race-adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for each rank category had CIs that spanned 1.0 (nonsignificant), but comparison of all officers as a group with all enlisted men as a group yielded an OR of 2.11 with a CI of 1.48-3.01 (significant). Moreover, by grouping the field-grade and general officers together as "senior officers" (37 total) and comparing that group with all of the others (a total of 193 enlisted men and company-grade officers), an OR of 3.30 and a CI of 1.99-5.45 (also significant) was obtained.	The findings of this paper are questionable for various reasons. First, the absence of data on the various occupational titles provided no basis for analyzing the validity of the results reported. Second, use of the registry of "overexposed" people to develop an exposure/job matrix is questionable because presumably each such incident was investigated to determine whether the person actually had been overexposed, and if so, for how long and with any health consequences. Also, such incidents are rare and more likely to occur in a few occupational titles.

TABLE 29N: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Grayson and Lyons (1996)	Studied was the incidence of various types of cancer in U.S. Air Force pilots and in Air Force personnel who had flown as crew during 1975-1979.	The comparison groups were non-flying Air Force officers and subjects in the NCI Surveillance, Epidemiology, and End Results (SEER) program.	Tabulated were the incidences of cancers of various types and body sites. For all cancer sites, the SIR was 1.19 with a 99% confidence interval (99CI) of 1.03-1.36 for the air crew group relative to the SEER group, and the risk ratio was 1.31 with a 99CI of 1.11-1.54 relative to non-flying officers. Contributing to the significant SIR were an excess of skin cancer with a SIR of 1.62 and a 99CI of 1.20-2.13 (primarily malignant melanoma) and of cancers of the urinary system with a SIR of 2.03 and a 99CI of 1.19-3.22. Those for the remaining cancer sites listed were nonsignificant, except for paucity of Hodgkin's disease, with a SIR of 0.51 and a 99CI of 0.23-0.99.	Specifically known carcinogenic agents or those presumed to be carcinogenic (such as RFEMF) that may have contributed to the few cancer excesses found were not discussed by the authors. Thus, their findings provide neither positive nor negative evidence for a relationship between cancer and RFEMF exposure.
Cox et al. (1982)	Occupational exposure levels from RFEMF heat sealers, for compliance with safety guidelines.	These authors measured the electric and magnetic fields at frequencies in the range 18-31 MHz at stations with 82 dielectric heat-sealer operators in 13 U.S. facilities.	Found was that 55% of the operators were being exposed to levels exceeding the maxima permitted in the OSHA (1978) guidelines [derived from ANSI (1974)].	Neither Cox et al. (1982) nor Joyner and Bangay (1986) discussed any specific health problems of the workers operating the sealers surveyed. The measurements are cited for their relevance to the study by Lagorio et al. (1997).
Joyner and Bangay (1986)		These authors surveyed the electric and magnetic fields from 101 dielectric heaters operating at 13.56, 27.12, or 40.68 MHz in Australia during 1982 and 1983.	Their results showed that 39% of the heaters were exposing the operators to levels that exceeded the Australian guidelines by factors between 1 and 10, and that the levels of another 23% exceeded those guidelines by factors greater than 10.	The numbers of subjects in the Bini et al. (1986) study were too few to ascribe credence to their findings, but was cited in Lagorio et al. (1997).
Bini et al. (1986)		Measured were the fields in a room containing 67 sealers (mostly at 27.12 MHz) for making thermal seams in various plastic-sheet articles in Italy.	The electric field near most of the units exceeded the levels allowed in Italian exposure guidelines. Mentioned was that 63 female workers had been interviewed for possible health effects, of which 30 had been exposed, 11 partially exposed (those not directly or continuously working at sealers), and 22 unexposed controls. The exposed group reported subjective complaints of eye irritation and upper-limb paresthesia (abnormal sensations such as burning, prickling), with statistically significant incidence relative to the other groups. The laboratory tests given all three groups showed no compromise of their general health.	

TABLE 29O: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

4 RFEMF AND CANCER IN NON-HUMAN MAMMALS

Many studies were carried out on various mammalian and non-mammalian species toward determining whether exposure to RFEMF *in vivo* or *in vitro* can result in mutations, cancer, or cause other damage to chromosomes or the genes therein. The fruit fly (Drosophila *melanogaster*) and several microorganism species have been used in standard tests for mutagenesis or carcinogenesis of various chemical or physical agents, because the short life spans of such species permit the study of many generations, well-characterized mutation-prone strains are available in large numbers, and baseline data exist on such strains for various agents. Carcinogenesis and mutagenesis have been found to be correlated in some aspects. For example, agents found to be mutagenic for the bacterium Salmonella *typhimurium* are also likely to be carcinogenic. However, not all mutagenic effects are possible indicators of carcinogenic effects and, conversely, not all carcinogenic effects are necessarily due to mutagenesis. Also sought were potential toxic effects on genes in cellular and subcellular preparations derived from animals exposed to RFEMF *in vivo* and from tissues exposed to RFEMF *in vitro*.

4.1 CANCER INDUCTION AND PROMOTION IN MAMMALS (*IN VIVO*)

As described previously, possible associations between chronic RFEMF exposure and cancer incidence were reported in various epidemiologic studies. In this section, studies specifically directed toward determining whether exposure of live animals to RFEMF induces or promotes cancer are discussed.

In an early study by Prausnitz and Susskind (1962), they exposed 200 male mice in groups of 10 for 4.5 minutes a day, 5 days a week, for 59 weeks to 9.3-GHz pulsed RFEMF (2-μs pulses, 500 pps) at 100 mW/cm^2 average power density. Such exposure caused a mean body-temperature rise of 3.3°C. Based on a prolate-spheroidal model of a mouse (Durney et al., 1978, pp. 97-99), the SAR is estimated to have been about 45 W/kg. The body-temperature rise sufficient to cause death in half the animals (LD_{50}) was 6.7°C, attained in 12 minutes at 100 mW/cm^2. Thus, exposure for 4.5 minutes at this power density was sublethal. Controls consisted of 100 sham-exposed mice.

Among the results at necropsy were liver abscesses in some mice, but because some tissues were lost by autolysis, the relative incidence of such abscesses in RFEMF-exposed and control mice could not be determined. Another finding was that the death rate was greater in the sham-exposed than in the RFEMF-exposed mice: At completion of the exposure series, 50% of the control mice and 65% of the RFEMF-exposed mice were still alive. The deaths in both groups were attributed to a pneumonia infection introduced accidentally into the colony during the experiment, and the authors suggested that the better survival of the RFEMF-exposed mice was due to the protective effect of the daily rise in temperature ("fever") induced by the RFEMF. The explanation is plausible, but not proven; RFEMF *in vivo* can have effects on the immune system.

These authors reported that some of the mice developed leukosis (also spelled leucosis), and that the incidence of leukosis was greater in the RFEMF-exposed than in the control mice. This was a real effect but its interpretation by the authors was probably faulty: In the paper, they described leukosis as a "cancer of the white blood cells", but leukosis is defined in dictionaries of medicine and pathology as an abnormal rise in the number of circulating white blood cells irrespective of the cause, rather than a form of cancer. Such dictionaries give detailed definitions of various types of leukemia, which indeed are cancers of the circulatory system. Various factors can give rise to leukosis, including stress, disturbances of the endocrine system, and infections such as the pneumonia in the colony noted above, which may have caused the observed liver abscesses.

Two other points should be considered as well. First, the incidence of leukosis in the RFEMF-exposed mice relative to the controls was higher but their percentage of survivors was also greater, a finding that would be considered unusual for most forms of leukemia in the mouse. Second, the incidence of leukosis in the RFEMF-exposed mice was higher during, but not following, exposure, with the implication that spontaneous remission of the "cancer" had occurred after the RFEMF exposure series was completed.

For true cancer, such remission would be considered quite improbable. Overall, the data from this study do not provide evidence as to whether or not chronic exposure of animals to RFEMF induces any form of cancer.

The conclusion above is supported by a reanalysis of the primary data of Prausnitz and Susskind (1962) by Roberts and Michaelson (1983), who noted that the results had been presented without statistical analyses. They restated the data as follows:

The first sacrifice, performed 7 months into the study, consisted of 10 mice of the RFEMF group and 5 of the control group. The second sacrifice, done at 16 months (1 month after completion of exposure), consisted of 20 RFEMF mice and 10 control mice. At 19 months (4 months after exposure completion), the surviving mice, which consisted of 67 RFEMF mice and 19 control mice, were all sacrificed. The respective totals of sacrificed RFEMF and control mice were 103 of 200 (52%) and 66 of 100 (66%). The remaining 97 RFEMF and 34 control mice had died at various times during the study. Of these, 60 of the RFEMF group and 40 of the control group were necropsied; the remainder had suffered excessive autolysis.

The incidences of leukosis in the first groups sacrificed had not been reported. In the second groups sacrificed, 6/20 (30%) of the exposed mice and 1/10 (10%) of the control mice had been reported as having leukosis. Roberts and Michaelson indicated that the difference was nonsignificant ($p>0.05$ by chi^2 analysis). In the third groups sacrificed, the numbers were 12/67 (18%) and 4/19 (21%), also a nonsignificant difference. By contrast, 67/200 (34%) of the exposed mice and 19/100 (19%) of the control mice had been alive just before the third sacrifice, a significant difference ($p<0.01$, chi^2 analysis). They concluded that the report "does not support a link between exposure to microwave radiation and the development of neoplasia", and they stated: "Thus, an equally plausible case could be made for the concept that the study by Prausnitz and Susskind (1962) demonstrated microwave-induced beneficial, rather than detrimental effects."

Skidmore and Baum (1974) tried to determine whether rapid changes in electric and magnetic fields would induce injuries in biological systems with high cell-turnover rates. They exposed male and female rats and male mice to electromagnetic pulses (EMP) continuously for 38 weeks (except for 2 hours a day on weekdays), totaling 100 million pulses. An EMP simulator was used, consisting of a parallel-plate transmission line fed by a pulse generator, with room for placing 200 nonmetallic cages between the plates. Each pulse had a rise time of 5 nanoseconds, a fall time of 550 nanoseconds, and a peak electric field of 447 kV/m; the pulses were delivered at 5 pulses per second. (Because of the short duration and shape of EMP, its broad RFEMF-frequency spectrum, and the low pulse-repetition rate, endeavors to calculate and use the equivalent free-space average power density and/or SAR for comparison with levels in other studies are not appropriate.)

One part of the study was to ascertain whether exposure to EMP altered the concentration or proliferative capacity of bone-marrow cells (the latter assessed from concentrations of mitotic cells). Dependence of these effects on exposure duration were determined by assaying them in six each of exposed and unexposed male rats after every two weeks of exposure. The results for both groups showed variations with time in numbers of nucleated bone-marrow cells and of mitotic rubricytes and myelocytes, but the differences in mean values between exposed and unexposed rats at corresponding times were not significant. Bone-marrow cells were arrested in metaphase and examined for chromosomal aberrations. In 2,000 cells from 40 rats exposed for 38 weeks, there were only 2 aberrations; the results were the same for 2,000 cells from 40 control rats. Blood samples taken from five each of the exposed and unexposed rats in the bone-marrow assays were given standard blood-chemistry assays. The rats were also given postmortem and histological examinations. None of the differences between groups in any of the blood-chemistry parameters were significant, and there was no RFEMF-associated pathology.

In another part of the study, blood samples (0.2 ml) were obtained on alternating weeks from two exposed groups of 10 male rats and from unexposed groups, and the concentrations of RBC, WBC, neutrophils, lymphocytes, reticulocytes, and platelets were determined. Assayed was incorporation of

Fe^{59} (a radiotracer) into newly formed erythrocytes in 30 each of exposed and unexposed rats. The Fe^{59} was injected into the tail vein of 6 rats each at 0.25, 1, 7, 14, and 21 days from the start of EMP exposure and in equal numbers of unexposed rats.

The graphs of reticulocyte concentration versus time for the exposed and unexposed groups showed that the counts for the exposed groups were almost always higher than for the control groups at corresponding times, with many of the differences statistically significant. However, with a few isolated exceptions, there were no consistent differences between the groups in the concentrations of circulating erythrocytes or in Fe^{59} uptake, thereby indicating no increase in RBC production. On the other hand, although the platelet counts for the exposed and control groups did not significantly differ initially, the counts for the exposed groups were almost always significantly lower than for the unexposed groups during most of the 38-week exposure period. The authors noted, however, that the consistently lower platelet concentration was within the normal concentration range and that it "represents no apparent functional problem". With a few isolated exceptions, there were no significant differences between the groups in circulating WBCs, neutrophils, or lymphocytes.

Noteworthy in the positive findings above is that the mean reticulocyte count for the exposed group before the start of exposure was higher than for the controls, and that the shapes of the reticulocyte graphs for the two groups were similar for about the first third of the 38-week exposure period. These may be an indication that uncontrolled non-RFEMF factors were present.

The authors also exposed five pregnant rats to 7 million pulses during 17 days of gestation, with five unexposed pregnant rats as controls. On completion of exposure, the fetuses were removed, fixed, and examined for gross abnormalities. None were found. Twenty female rats were continuously exposed and, with 20 controls, were observed for possible development of mammary tumors. At age 1 year (after exposure to 10 million pulses), no mammary tumors were found in either group.

Last, 50 male AKR/J mice, a strain known to be prone to spontaneous development of leukemia between 6 and 12 months of age, were exposed to the EMP, with 50 mice as controls. At the end of 33 weeks (86 million pulses), the 42 surviving exposed mice and the 24 surviving control mice were euthanized and examined histopathologically for the presence of leukemia. Nine of the exposed mice (21%) and 11 of the control mice (46%) were leukemic, indicating that the exposure to EMP was not involved in leukemia development. There were no significant differences between groups in mean thymic or splenic weight or WBC count.

Baum et al. (1976), noting that the study above encompassed only about half the normal life span of the rats, performed a similar study (same EMP characteristics) with male and female rats but for 94 weeks (except for 1 hour a day on weekdays). The exposures were started at age 4 months, for a total of 250 million pulses. The reticulocyte count during the early part of the exposure was higher in the exposed than in the control group, as in the previous study, but the differences between groups for the last 25 weeks were nonsignificant. This was also true for the platelet count. There were also no significant differences between the groups in the other biological endpoints previously studied.

Preskorn et al. (1978) performed two studies of effects of exposure to hyperthermal 2.45-GHz RFEMF on tumor growth and longevity in mice implanted with lymphoreticular cell sarcoma. In the first study, four pregnant CFW mice were exposed to the RFEMF in the multimode microwave cavity of Justesen et al. (1971) at an average whole-body dose rate [SAR] of 35 W/kg for 20 minutes daily during gestation days 11-14. Selected from the four resulting litters were 24 weanlings of both sexes. Four other pregnant mice were sham-exposed, and 24 weanlings were similarly selected therefrom. On postpartum day 16, all 48 pups were injected subcutaneously with a sterile homogenate of tumorous tissue that contained avian fast reticuloendothelial T virus (0.6-1.2 cells in 0.1 ml of injectate). A 2x2 factorial experimental design was used, with 12 mice each assigned to one of four conditions:

Group 1--Mice sham-exposed in utero on gestation days 11-14 and sham-exposed postnatally for 20 minutes per day on days 19 through 54.

Group 2-- Mice sham-exposed in utero but RFEMF-exposed postnatally for 20 minutes per day on days 19 through 54 (36 exposures).

Group 3--Mice similarly RFEMF-exposed in utero but sham-exposed postnatally.

Group 4--Mice similarly RFEMF-exposed both in utero and postnatally.

Groups of four (or more) dams were concurrently RFEMF- or sham-exposed, as were groups of four weanlings each. By calorimetry, the whole-body SAR per mouse was stated to have ranged from 33 to 38 W/kg with an average of 35 W/kg, which yielded an average total dose per 20-minute treatment of about 42 joules per gram. Not clear, however, is whether those values were for the dams, for the pups, or both, and to what extent the values may have changed with time as the weights of the mice increased. Colonic temperatures, sampled immediately before and after RFEMF exposure, averaged respectively 37.74°C and 39.98°C (a mean rise of 2.24°C).

After implantation of the homogenate, each weanling was examined daily both visually and by palpation until postpartum day 93. At that time, they were euthanized and given histopathologic examination, including tissues from the implantation site by light microscopy, and macroscopic and microscopic evaluation of organ systems, all performed under blind control. The authors noted that all tumors were identified as sarcomas, and that no tumor found microscopically were had not been detected by palpation.

The results of this first experiment were displayed in Figure 1 of the paper as bar graphs of the numbers of induced sarcomas in each group. The numbers of sarcomas in Group 1 (prenatal and postnatal sham exposure) and Group 2 (prenatal sham exposure and postnatal RFEMF exposure) respectively were 6 (in 12 mice) and 5, a nonsignificant difference with a mean of 5.5. The authors indicated that half of those in Group 1 exhibited tumors, an induction rate comparable to that reported by Nielsen (1969) for commercially available CFW mice, and that only 5 of the 12 mice in Group 2 had tumors despite postnatal RFEMF exposure. [Presumably all tumor-afflicted mice had only 1 tumor each.] The results for Groups 3 (prenatal RFEMF exposure and postnatal sham exposure) and 4 (both prenatal and postnatal RFEMF exposure) were only 2 and 1 tumor, respectively, with a mean of 1.5. The authors, noting an apparent association between reduction of tumor incidence and postnatal RFEMF exposure, stated that by 2-tailed 2x2 chi^2 test with Yates's continuity correction, the probability of the association occurring by chance is less than 0.05. They also remarked that there was evidence of tumor regression in one mouse each in Groups 3 and 4; in both mice, the growths diminished in size and only the residuum of an inflammatory reaction was evident at necropsy time.

In the second experiment, larger numbers of gravid CFW mice were used, all hyperthermal treatments were done in utero (at 35 ± 3 W/kg), and all mice were observed for longevity for three years. Specifically, 10 primigravid mice were exposed to the RFEMF for 20 minutes each on gestation days 11, 12, 13, and 14, and 84 weanlings were selected randomly for gender representation except that they be of normal body masses and free of externally observable defects. Similarly, 8 other primigravid mice were sham-exposed, and 60 of their weanlings were selected.

The 144 weanlings were implanted with the T virus homogenate on day 16 postpartum, after which groups of 5 or fewer mice were segregated by gender and placed in cages in a windowless mouse vivarium with food and water continuously available. The cages of both groups were maintained in close proximity and at equivalent heights and locations. Light from fluorescent lamps was cycled on from 6 am to 6 pm daily. The mice were checked daily by an investigator or by an animal-care technician for 36 months. A mouse was regarded as tumor-bearing only if a palpable growth at the implantation site persisted to time of mortality; confirmed tumors were dated from the first day of appearance.

The results were plotted in Figure 3 of the paper as percentage of mice with tumors versus months after homogenate implantation for the RFEMF-exposed and sham-exposed mice. For the sham-exposed mice, the curve rose steeply to about 37% at 2.5 months (the time at which the mice of the first experiment were necropsied) and plateaued at 40% for the remainder of the examination period. By

contrast, the curve for the RFEMF-exposed increased approximately linearly to only 15% at 2.5 months and it continued to rise similarly to a plateau of 46% between 5 and 6 months. By chi^2 test, the difference between the values at 2.5 months was highly significant ($P<0.01$), but the difference between the curves subsequently narrowed to where the 6% difference between the plateau values was nonsignificant ($P>0.1$).

Also plotted (in Figure 4) were percentage of surviving mice versus age in months for the two groups. Starting at age about 2+ months, survival of the sham-exposed mice diminished from 100% to about 50% at about 6 months, to about 10% at 9 months, and to almost 0% at 12 months. However, survival of the RFEMF-exposed mice remained at 100% until 3+ months, diminished to about 77% at 6 months, decreased to about 20% at 9 months, and linearly more slowly to almost 0% at 18 months. The authors again noted that regression of tumors had occurred in some RFEMF-exposed mice.

The average survival time for mice that did not develop tumors was also longer for the RFEMF-exposed than the sham-exposed mice. Specifically, at age 24 months, when 50% of the sham-exposed mice had survived, 67% of the RFEMF-exposed mice had survived, with the two curves converging to near 0% at about age 36 months.

The authors ascribed the better outcomes for the RFEMF-exposed mice to an enhanced immunocompetency from moderate short-term elevation of fetal- and perhaps maternal body temperature (the latter by about 2.24°C) from the RFEMF exposure of the dams, but considered the findings provocative but inconclusive without histological verification. These survival results seem analogous to those of Prausnitz and Susskind (1962), but the RFEMF hyperthermia in the latter study apparently served to aid the mice in fighting pneumonia infection in the mouse colony. Preskorn et al. (1978) did not discuss whether there were any health problems other than tumors in their mice.

Berman et al. (1980), in the first of three experiments, used 12 male rats that previously had been exposed to 2.45-GHz CW RFEMF at 5 mW/cm^2 for 4 hours a day from gestation day 6 to age 90 days, and 12 rats that had been similarly sham-exposed. [The 24 rats were 90-day-old survivors in a study of possible effects of RFEMF on lymphocytes by Smialowicz et al. (1979)]. The mean SARs ranged from 4.7 W/kg for ages 1-5 days to 0.9 W/kg for ages 31-40 days and were less than 0.9 W/kg for rats older than 40 days.

In experiment 2, 14 young adult rats were exposed 5 hours a day for 5 days to 2.45-GHz CW RFEMF at 10 mW/cm^2, and 16 rats were sham-exposed. In experiment 3, 6 rats were exposed 4 hours a day, 5 days a week, for 4 weeks to 2.45-GHz CW RFEMF at 28 mW/cm^2, and 6 rats were sham-exposed. The mean SARs were not determined in experiments 2 and 3, but were estimated as 2.0 and 5.6 W/kg, respectively. Rectal and intratesticular temperatures were monitored in anesthetized mature rats during 90 minutes of exposure at 28 mW/cm^2 and during sham exposure.

Development of mature sperm in rats takes about 70 days. Starting on the third day after completion of treatment, each male of experiment 1 was housed with a pair of 90-day-old virgin females for three separate weeks of breeding spaced about a week apart, a total period encompassing the early stages of spermatogenesis. Those of experiments 2 and 3 were similarly bred for weekly periods, but extending respectively to 72 and 80 days after treatment cessation, encompassing essentially all stages of spermatogenesis. The males of experiments 2 and 3 were bred for one week prior to exposure, to assess their reproductive abilities. Results for those that had not sired at least one normal pregnancy with at least one fetus during the pretreatment or the first post-treatment breeding were excluded. (Two RFEMF-exposed males in experiment 2 were excluded.)

On day 10 following the weekly breedings, those females were euthanized and examined for pregnancy, number of live and dead conceptuses, and number of corpora lutea. The preimplantation loss was calculated by subtracting the total number of conceptuses (live + dead + resorbed) in each litter from the number of corpora lutea. The authors noted that if the preimplantation loss for an RFEMF group is significantly larger than for the corresponding sham group, it may indicate embryonic death due to mutagenic alteration of sperm.

During the week after the final breeding, the males in experiments 2 and 3 were euthanized and their bodies and organs were weighed. Both caudal epididymes were removed from the rats of experiment 2, placed in warm saline, and cut into small pieces to liberate the sperm from the lumena. The suspensions were diluted and fixed with neutral formalin, and the relative sperm concentration in each was determined by particle counting. Smears were taken prior to fixation, and the sperm were fixed and stained; 200 cells were examined and the live cells were counted.

Only data from RFEMF and sham groups for the same breeding period and experiment were compared. Appropriate statistical techniques were used to compare groups with regard to the various endpoints. Litter values were used in analyzing the numbers of live and dead conceptuses and postimplantation losses.

In experiment 1, the only significant difference was in the mean number of dead fetuses per litter, which was higher in the females mated with sham-exposed males than in those mated with RFEMF-exposed males, and only for one of the breeding weeks. Clearly, this difference was not RFEMF-induced. Moreover, in experiment 2, none of the differences in mean numbers of pregnancies or live, dead, and total fetuses per litter or preimplantation loss per litter were significant.

In experiment 3, which involved breedings during days 3-9, 17-23, 31-37, 45-51, 59-65, and 72-78 after sham- or RFEMF exposure of the males, there were only two significant differences. Eleven of the 12 females bred with sham-exposed males 3-9 days after treatment were pregnant, but only 6 of the 12 females bred with RFEMF-exposed males were pregnant. For the breeding period 17-23 days, the mean number of live fetuses per litter for 11 of the 12 females of the sham group found pregnant was higher than for the 12 pregnant females of the RFEMF group. The corresponding mean number of dead fetuses per litter did not differ significantly, but the total number of fetuses for the sham-exposed group in that breeding period was significantly higher than for the corresponding RFEMF-exposed group.

For the corresponding groups of RFEMF-exposed and sham-exposed males in experiments 2 and 3, there were no significant differences in mean body weights or weights of testes, liver, or adrenals. Also not significant were the differences in relative concentration of epididymal sperm or percentage of live sperm between groups in experiment 2.

During 90 minutes of sham exposure of anesthetized rats, both the mean rectal temperature and the mean intratesticular temperature decreased, from 38.8°C to 36.5°C and from 34.6°C to 32.1°C, respectively. For those exposed at 28 mW/cm^2, the mean rectal temperature increased from 38.4°C to 40.9°C and mean intratesticular temperature from 33.9°C to 37.5°C.

In summary of this investigation, no evidence was found for an increase of dominant lethal mutations induced by RFEMF at power densities up to 28 mW/cm^2 (SARs up to 5.6 W/kg). Regarding male fertility, the authors remarked that only under the most severe exposure regimen (experiment 3) was there any hint of a deleterious effect: Only 50% of the dams bred to exposed males 3-9 days after treatment showed pregnancies. Presumably this temporary sterility was associated with the significant increases in rectal and intratesticular temperatures.

McRee et al. (1981), remarking that analysis of induction of sister chromatid exchange (SCE) is a sensitive technique for assaying genetic damage of mutagens and carcinogens, used this technique to determine whether RFEMF is mutagenic in mice. A horizontal circular array of 12 ten-week-old mice in individual Styrofoam cages was exposed from above to far-field 2.45-GHz CW RFEMF at 20 mW/cm^2 in an anechoic chamber at 22°C and 55% relative humidity. The exposures were for 8 hours a day (4 hours each in the morning and afternoon) for 28 days.

The authors determined the SARs for several mouse configurations and orientations relative to the RFEMF by measuring deep colonic temperatures in dead mice during exposure at 20 mW/cm^2 and using the heating curve during exposure and the postexposure cooling curve. The SARs ranged from 15.6 to

26.8 W/kg, but the SARs for live mice in the experiment were near the latter value because during most of each exposure, the mice were curled up and essentially parallel to the electric vector.

Two other groups of 12 mice each were used as controls. One group was sham-exposed in the RFEMF chamber under conditions otherwise similar to those for the RFEMF group; the other group was kept in home cages that were placed within a second environmental chamber identical to the one used for RFEMF- and sham exposure and were not handled.

The SCEs in the bone marrow of the femurs of the mice were determined immediately after completion of 28 days of exposure. At least 15 cells per mouse were examined. The mitotic index was scored as the proportion of metaphase cells in a sample of more than 1,000 bone marrow cells from each mouse. The results for only three mice of each group were presented; the remaining mice were eliminated primarily because the counts of cells per sample were inadequate, a feature inherent in the specific type of assay used. The results were tabulated.

The mean of means for the RFEMF, sham, and control groups were comparable, about 3 SCEs per metaphase cell, an indication that the RFEMF exposure did not significantly affect sister chromatid exchanges. In addition, analysis of the mitotic-index values showed that the RFEMF had no significant effect on the rate of proliferation of bone-marrow cells.

A study by Saunders and Kowalczuk (1981) had shown that exposure of the rear halves of anesthetized mature male mice to 2.45-GHz RFEMF in a waveguide system at half-body SARs of 43 W/kg or higher for 30 minutes severely reduced sperm production during the heat-sensitive stages. Saunders et al. (1983) therefore sought to determine if such exposure was also mutagenic for male germ cells. Four groups of six sexually mature male mice each were anesthetized, and the rear halves of their bodies were exposed to 2.45-GHz RFEMF for 30 minutes at 43.4 W/kg (half-body SAR) in the waveguide system used previously. For each RFEMF group, a group of six mice was similarly sham-exposed. For a positive control, 11 mice (groups of 5 and 6) were exposed to 170-keV X-rays, with appropriate sham exposure groups. Mean rectal temperatures measured immediately after RFEMF exposure and sham exposure were 41.5°C and 34.1°C, respectively.

Following treatment, each male was caged with two sexually mature virgin female mice for seven days, after which the males were placed in fresh cages and mated with a second batch of females. The procedure was repeated until the males had been mated for 8-10 weeks. The female mice were euthanized 14 days after mating if a vaginal plug was present or 18 days after caging if not. When uterine implants were present, large and small deciduomata, indicative of development that was halted by early embryo death, were counted. Also counted were the corpora lutea, live embryos, and late fetal deaths.

The percentage of females rendered pregnant by mating with the RFEMF-exposed males was comparable to the percentage of those mated with sham-exposed males during the first two mating weeks, but it significantly diminished for weeks 3 through 8 and rose to comparable values again by week 10. For each week, the numbers of corpora lutea per pregnant female of the RFEMF and sham groups were comparable. However, the number of total implants for the RFEMF groups diminished by week 5 to a minimum of about half that for the corresponding sham group and then recovered by week 10. On the other hand, the percentages of live implants to total implants for the RFEMF and sham groups were comparable each week. Analyses of these results by a sequence of five successive hierarchical models to assess the separate contributions of various possible factors to the overall responses confirmed the statistical significance of the differences above.

Thus, the results of Saunders et al. (1983) showed no evidence of RFEMF-induced dominant lethal mutagenic effect. The diminution of total implants was ascribed to decreased male fertility, in consonance with the previous findings (Saunders and Kowalczuk, 1981).

Szmigielski et al. (1982) investigated whether exposure to RFEMF would: decrease the natural resistance of Balb/c mice to lung-cancer cells injected intravenously before exposure, increase the

incidence of breast tumors in female C3H/HeA mice (a strain known to have high spontaneous incidence of such tumors), and increase the incidence of skin cancer in male Balb/c mice that were locally depilated and painted with the chemical carcinogen 3,4-benzopyrene (BP).

RFEMF exposures were to far-field 2.45-GHz RFEMF at 5 mW/cm^2 (SAR 2-3 W/kg) or 15 mW/cm^2 (6-8 W/kg). For exposure, 40 mice were placed in 4 polymethacrylate cages holding 10 mice each. Other groups of mice were sham exposed. The exposures were for 2 hours a day, 6 days a week, for periods of 1 to 6 months. The temperature (22-23°C) and humidity (60-70%) within the exposure chamber were held stable by external ventilation.

As additional controls to normal (cage-control) and sham-exposed mice, groups of male Balb/c mice were grown for 1 to 8 months, starting at age 6 weeks, within cages 20x30x10 cm in size containing 20 transparent 5x6x10-cm compartments, with one mouse in each compartment. The authors (citing appropriate references) noted that growth under such confinement causes a chronic stress syndrome with aggressiveness.

In the lung cancer study, Balb/c mice were intravenously injected with sarcoma L1 cells. The mice were killed 14 days later, their lungs were infused with India ink in fixative, and the numbers of white neoplastic nodules (colonies originating from single cells) were counted. Based on pilot experiments, the concentration of sarcoma L1 cells used was 2x105 L1 cells (in 0.1 ml of saline), which yielded a mean control value of 2.8 ± 1.6 (SD) nodules per mouse.

RFEMF exposure of injected mice for 3 months at 5 mW/cm^2 produced 6.1 ± 1.8 nodules, whereas 10.8 ± 2.1 nodules were seen for exposure at 15 mW/cm^2, a significant difference. Injection of mice that had been grown in confinement for 3 months showed 7.7 ± 2.0 nodules, a mean comparable to that for exposure at 5 mW/cm^2. Smaller but significant differences were seen after RFEMF exposure or confinement for 1 or 2 months.

In the breast-cancer investigation of C3H/HeA mice, groups of 40 mice each were exposed to RFEMF from age 6 weeks up to age 12 months, and each mouse was checked every 2 weeks for palpable breast tumors. The cumulative numbers of mice with discernible tumors and their survival times were tabulated. By regression analysis, the results were summarized in terms of CDT_{50} (mean cancer development time in 50% of the mice) and MST_{50} (mean survival time of 50% of the mice). The CDT_{50} values were 219 days for 15 mW/cm^2, 255 days for confinement-stressed mice, 261 days for 5 mW/cm^2, 297 days for sham exposure, and 322 days for cage controls. Thus, the CDT_{50} values for 5 mW/cm^2 and confinement stress were comparable, and were between those for 15 mW/cm^2 and the cage controls. The results for MST_{50} were analogous.

Table 30, adapted from Table 1 of the paper, showed the cumulative numbers of mice with tumors at 4, 6, 8, and 10 months:

TABLE 30: CUMULATIVE NUMBERS OF MICE WITH TUMORS
[Szmigielski et al. (1982)]

Treatment; Tallied At:	4 months	6 months	8 months	10 months
Cage control	0	0	2	16
Sham-exposed	0	0	3	14
RFEMF at 5 mW/cm^2	0	3	18**	32**
RFEMF at 15 mW/cm^2	1	11**	26**	37**
Confinement stress	0	2	16**	31**

**$p < 0.01$ relative to cage controls.

Sham exposure had no significant effect on the numbers of mice with tumors, but exposure at 5 mW/cm^2 and confinement stress yielded comparable increases in the numbers of mice affected. Exposure at 15 mW/cm^2 yielded significantly higher increases.

No increase in rectal temperature was seen at 5 mW/cm^2 (2-3 W/kg), but the authors noted that such SARs exceed the basal metabolic rate of the mice. No rectal-temperature increase was seen at 15 mW/cm^2 (6-8 W/kg) either, but the authors suggested the possible existence of "hot spots" within the mice. At the higher level, the RFEMF must have increased the heat stress on the mice considerably. Because confinement-stress alone was shown to increase tumor incidence (presumably an effect on the immune system as mediated by the endocrine system), it seems plausible that the heat from the RFEMF (also affecting the immune system) caused the increases in tumor incidence rather than existence of any intrinsic carcinogenic properties of the RFEMF.

To evaluate the effects of BP alone in the skin-cancer experiments, 40 six-week-old male Balb/c mice were depilated over a 1-cm^2 area of skin, and the areas were painted with BP (in a solvent) every other day for 5 months. Controls were similarly depilated but painted only with the solvent. Cancer development was scored by histopathologic examination on a subjective 7-grade scale from 0 to 6. At a score of 4, small papillomas were found microscopically to contain cancer cells, so mice with scores of 4-6 were regarded as having skin cancer and those with scores 1-3 as having precancerous skin lesions. Skin cancer occurred within 7-10 months in more than 85% of those treated with BP.

Table 31, adapted from Table 2 of the paper, shows the cumulative numbers of mice with skin cancer (scores 4-6) for cage controls (5 mice per cage), sham exposure, RFEMF exposure at 5 mW/cm^2, or confinement stress for 1 and 3 months before BP treatment.

TABLE 31: CUMULATIVE NUMBERS OF MICE WITH SKIN CANCER FROM EXPOSURE TO RFEMF PRIOR TO BP TREATMENT
[Szmigielski et al. (1982)]

Treatment; Tallied At:	4 months	6 months	8 months	10 months
Cage controls	0	0	3	18
Sham-exposed	0	0	4	19
RFEMF at 5 mW/cm^2 (for 1 month before BP)	0	2	18**	27*
Confinement stress (for 1 month before BP)	0	3	16**	24
RFEMF at 5 mW/cm^2 (for 3 months before BP)	1	22**	29**	36**
Confinement stress (for 3 months before BP)	0	16**	25**	31*

*$p < 0.05$ relative to cage controls.
**$p < 0.01$ relative to cage controls.

As in the breast-tumor study, the numbers of mice affected by exposure at 5 mW/cm^2 or confinement stress were comparable. Not clear is whether similar experiments were done for exposure at 15 mW/cm^2 for 1 and 3 months before BP treatment.

About CDT_{50} values, control mice (treated only with BP) developed skin cancer (scores 4-6) in about 10 months (a CDT_{50} of 296 days). The results for the other treatments were obscure due to the order of their presentation, but presumably indicated that the CDT_{50} values for sham exposure, confinement stress, exposure at 5 mW/cm^2, or exposure at 15 mW/cm^2 for 3 months before BP treatment were respectively 272, 201, 171, and 171 days, with the latter three values significantly lower than for the control mice. Not clear is the lack of difference between the CDT_{50} values for 5 and 15 mW/cm^2.

Table 32, adapted from Table 3 of the paper, were the results for concurrent RFEMF exposure and BP treatment. The only corresponding CDT_{50} value indicated was 131 days for mice exposed at 15 mW/cm^2. However, the MST_{50} values were 331, 268, 237, and 165 days for cage controls, exposure at 5 mW/cm^2, confinement stress, or exposure at 15 mW/cm^2, respectively.

TABLE 32: CUMULATIVE NUMBERS OF MICE WITH SKIN CANCER FROM CONCURRENT RFEMF AND BP TREATMENT
[Szmigielski et al. (1982)]

Treatment; Tallied At:	4 months	6 months	8 months	10 months
Cage control	0	0	3	18
Sham-exposed	0	0	5	21
RFEMF at 5 mW/cm^2	2	13**	26**	31**
RFEMF at 15 mW/cm^2	1	12**	23**	32**
Confinement stress	9*	28**	33**	38**

*$p < 0.05$ relative to cage controls.
**$p < 0.01$ relative to cage controls.

Again, the RFEMF-induced increases in skin-cancer incidence were probably due to the heat stress rather than any postulated intrinsic carcinogenic properties of the RFEMF. In their discussion, the authors stated:

"It may be postulated that the differences in the appearance of tumors and the number [of] lung cancer colonies between animals irradiated with 2,450-MHz MWs at 5 and at 15 mW/cm^2 may be due to local thermal effects evoked at 15 mW/cm^2."

They also noted that 2.45-GHz RFEMF is close to the resonant frequencies for mice, that maximal absorption by humans at this frequency would be almost two orders of magnitude lower than for mice, and that RFEMF absorption by humans at their resonant frequencies (60-70 MHz) would be about 20% of the RFEMF absorption by mice at the latter's resonant frequencies. The authors stated: "Thus, the mouse model used in this study is of very limited value for concluding about the possible hazards from MW radiation in human subjects."

In a brief communication, Santini et al. (1988) discussed their results on whether low-level exposure of black mice (strain C57BL/6J) would have any effect on the development of B16 melanoma or survival times. They exposed one group of 15 female mice, 5 weeks old, to CW 2.45-GHz RFEMF at 1 mW/cm^2 (SAR 1.2 W/kg) for 6 daily sessions per week, each session for 2.5 hours a day, until death (up to 690 hours total). Another group of 15 female mice was similarly exposed to pulsed 2.45-GHz RFEMF at the same average power density. The pulsed RFEMF consisted of bursts of 10-μs spikes with 5 μs between spikes, and with each burst lasting 10 ms with 30 ms between pairs of bursts. A group of 15 female mice was sham-exposed as controls.

The two RFEMF exposure groups were exposed for 15 days, after which all three groups were subcutaneously implanted with 3 million (0.1 ml) melanoma cells. After inoculation, RFEMF- and sham exposure were continued until the mice in all three groups died. At periodic intervals during tumor development, the largest surface dimension of each tumor and its surface dimension at right angles thereto were measured with a gauge, and the product was taken as the tumor area. The results were shown in Figure 1 of the paper as bar graphs of mean tumor surface areas and standard deviations at time intervals of 9, 12, 16, 20, 23, and 27 days after inoculation. As would be expected, the mean tumor area for each group increased with time. At each time interval, the SD bars for the three groups overlapped, and the authors stated that by Student's t-test, there were no significant differences among the groups. However, no experimental data were presented, such as the numbers of mice afflicted in each group or the numbers of tumors in each mouse.

Mean survival times and SDs in days for the mice afflicted with B16 melanomas were presented in Table 1 of the paper. The results for the sham, CW, and pulsed groups respectively were 25.9 ± 6.2, 24.4 ± 7.9, and 26.6 ± 8.5. The authors indicated that they had analyzed the numbers of surviving and dead mice by chi^2 test and the survival times by Wilcoxon-Mann-Whitney U-test. No results were given for the chi^2 test, but the Wilcoxon-Mann-Whitney U-test on individual survival times showed no significant differences among the groups.

Although the findings of this study showed no significant differences among the RFEMR-exposed and sham-exposed mice in melanoma development and survival times, the credibility of those negative findings is vitiated to some degree by the absence of experimental data, even though the paper was a brief communication.

A study was performed at the University of Washington in which 100 male Sprague-Dawley rats were exposed throughout their lifetimes (up to 25 months) under specific-pathogen-free (SPF) conditions within individual cylindrical waveguides to 2.45-GHz RFEMF at average power density of about 0.5 mW/cm^2. One hundred rats were concurrently sham-exposed in identical waveguides. The whole-body SARs ranged from 0.4 W/kg for a 200-g rat to 0.15 W/kg for an 800-g rat. Evaluated were a wide range of biological endpoints (155) toward assessing possible effects of such chronic exposure. The findings were presented in nine technical reports (Chou et al., 1983b; Guy et al., 1983a, 1983b, 1985; Johnson et al., 1983, 1984; Kunz et al., 1983, 1984, 1985), and were discussed in a subsequently published paper by Chou et al. (1992).

For most of the 155 biological endpoints, no statistically significant differences were found between the RFEMF-exposed and sham-exposed rats. One general finding was that the median survival time of the RFEMF-exposed rats was somewhat longer than for the sham-exposed rats (688 versus 663 days). Another finding specifically relevant to this report were the incidences of lesions characterized in Table 2 of Chou et al. (1992) as either non-neoplastic or neoplastic, with the latter noted as benign (B), primary (P) or metastatic (M). Shown in that table were the organs or sites of occurrence, the specific types of lesions at each site or organ (for a total of 83 types), and the occurrences of B, P, and M for each lesion type in both the RFEMF-exposed and sham groups. The authors noted that a primary (P) lesion is one that occurs in only one organ system, whereas a metastatic (M) lesion may occur in many organ systems of a single animal resulting from a P lesion.

Regarding non-neoplastic lesions, chronic glomerulonephropathy (a non-inflammatory disease of the internal structures of the kidney) was the most frequent cause of death, but was found in fewer of the RFEMF-exposed than the sham-exposed rats. The differences in occurrence and severity of other non-neoplastic lesions between the two groups were not statistically significant.

The authors stated that the incidence of neoplastic lesions corresponded to that normally reported in the literature for Sprague-Dawley rats. Only two tumors were found in the rats younger than 12 months, and the incidence rose rapidly after age 18 months. As expected in the aging rats, the highest incidence of neoplasia was in the endocrine system. The incidence of benign pheochromocytoma (a vascular tumor in the adrenal medulla) was higher in the RFEMF-exposed rats than in the sham-exposed rats, but the difference was not statistically significant ($p=0.065$, by Fisher's exact test).

Overall without regard to age, primary malignancies were observed in 18 RFEMF-exposed rats but in only 5 sham-exposed rats. For each specific type of malignancy, the difference in incidence between the RFEMF-exposed and sham-exposed rats was not statistically significant. However, the collective incidences (18 versus 5 rats) without regard for the site or organ of occurrence differed significantly, based on the Mantel-Haenszel estimate of the odds ratio and the chi^2 test. For most of the 155 biological endpoints, no statistically significant differences were found between the RFEMF-exposed and sham-exposed rats. One general finding was that the median survival time of the RFEMF-exposed rats was somewhat longer than for the sham-exposed rats (688 versus 663 days). Another finding specifically relevant to this report were the incidences of lesions characterized in Table 2 of Chou et al. (1992) as either non-neoplastic or neoplastic, with the latter noted as benign (B), primary (P) or metastatic (M). Shown in that table were the organs or sites of occurrence, the specific types of lesions at each site or organ (for a total of 83 types), and the occurrences of B, P, and M for each lesion type in both the RFEMF-exposed and sham groups. The authors noted that a primary (P) lesion is one that occurs in only one organ system, whereas a metastatic (M) lesion may occur in many organ systems of a single animal resulting from a P lesion.

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Overall without regard to age, primary malignancies were observed in 18 RFEMF-exposed rats but in only 5 sham-exposed rats. The difference in incidence between the RFEMF-exposed and sham-exposed rats was not statistically significant for each specific type of malignancy but the collective incidences (18 versus 5 rats) without regard for the site or organ of occurrence differed significantly. These findings were based on the Mantel-Haenszel estimate of the odds ratio and the chi^2 test, with both statistical tests insensitive to the grouping of the data with respect to age at death. In their discussion, the authors stated:

"Although the overall difference in numbers of primary malignancies is statistically significant, the biological significance of this difference is open to question. First, detection of this difference required the collapsing of sparse data without regard for the specific type of malignancy or tissue of origin. Also, when the incidence of the specific primary malignancies in the exposed animals was compared with specific tumor incidence reported in the literature, the exposed animals had an incidence similar to that of untreated control rats of the same strain maintained under similar SPF conditions."

Fučić et al. (1992) noted that micronuclei of mammalian cells originating from chromatid fragments ought to be generally smaller than those from whole chromosomes. In this study, they assayed the sizes of lymphocyte micronuclei in 100-cell samples from three groups of humans occupationally exposed to X-rays, microwaves, or vinyl chloride monomer, characterized by the authors as "well known mutagenic agents". In the last two years, the X-ray group had been exposed to doses lower than 25 millisieverts (mSv) ["Sv", a unit of radiation absorption dose (rad) of 100 ergs per gram, also equivalent to the Gy]. Measurements at the workplaces of the group exposed to "pulsating microwaves" indicated that the subjects had been exposed to frequencies in the range 1.25-1.35 GHz at power densities that varied between 10 $\mu W/cm^2$ and 20 mW/cm^2. For those exposed to vinyl chloride monomer, the mean concentration in the working environment was 50 ppm, but it periodically reached 2000 ppm for about 4 hours a week.

Each group had been employed for about 15 years on average. All of the exposed subjects were non-smoking males in the age range 25-45 years. An unexposed group of non-smoking males of similar age distribution served for comparisons. The numbers of subjects in the groups or details about their specific occupations, related exposure levels, and durations were not given.

The surface areas of the micronuclei were measured with a Morphomat 10 and were divided into eight equal-size classes, each comprising an eighth of the difference between the smallest and largest micronuclei detected. The distributions of the three exposed groups and the control group were shown as histograms of the numbers of micronuclei versus their size class.

The number of micronuclei for the microwave group was highest in the smallest size class (33), was nearly as high in the next higher size class (30), was much lower in the third size class (9), and decreased monotonically in the remaining classes. By comparison, the numbers for the controls in the smallest, next smaller, and third smaller size classes were 11, 27, and 16 micronuclei, respectively. The corresponding results for the X-ray group were 13, 39, and 23 micronuclei, and those for the vinyl chloride group were 29, 19, and 17. The differences between each exposed group and the control group were said to be significant, as were some of the differences among the exposed groups. The authors used the latter findings to characterize the differences among the exposed groups in the type of damage caused.

Specifically, they indicated that X-rays and microwaves were preferentially clastogenic (causing chromosome breaks), and that vinyl chloride monomer was aneugenic (causing chromatid breaks).

As noted above, details about the occupational aspects of the exposure and control groups were not given. Also not presented were the numbers of 100-cell samples assayed from each subject or the variances thereof (if any), or any specific data on the statistical treatment given other than that the nonparametric Kolmogorov-Smirnov test was used. Consequently, any validity of these findings is questionable.

Salford et al. (1993) indicated that the rat glioma cell line RG2 gives rise to glioma-like tumors when injected into the brains of Fisher 344 rats. They stated that when at least 1,000 RG2 cells are inoculated, well delineated tumors develop in 100% of their animals within three weeks, at which time the untreated tumors have reached diameters of about 3-6 mm. The authors sought to determine whether exposure of rats to RFEMF after such brain inoculation would promote the growth of the brain tumors initiated by the RG2 cells.

Seventy-four Fisher 344 rats of both sexes were stereotaxically injected with 5,000 RG2 cells in nutrient solution into the head of the right caudate nucleus. Starting on day 5 after inoculation, 37 of the rats were exposed without anesthesia to 915-MHz CW RFEMF; or to 0.57-ms pulses of 915-MHz RFEMF at repetition rates of 4, 8, 16, or 217 pps; or to 6-ms pulses of 915-MHz RFEMF at a repetition rate of 50 pps. The exposures were done in well-ventilated TEM cells, with identical TEM cells used for sham exposure. The whole-body SARs were tabulated; they ranged from 0.0077 W/kg for the 0.57-ms pulses at 4 pps to 0.4 W/kg for 217 pps, and were 1.00 W/kg for 6-ms pulses at 50 pps and 1.67 W/kg for CW. The exposures were for 7 hours a day, 5 days a week for 2 or 3 weeks, with a 30-minute break each day for the feeding and stretching of legs after 4 hours of exposure. None of the rats showed signs of unrest during the treatments, and they returned spontaneously to the TEM cells after the lunch breaks.

The rats were treated in groups of 2 or 4; each rat selected for RFEMF exposure was matched with a control rat for sham exposure. The rats received between 9 and 15 treatments. Their rectal temperatures were measured with an optical thermometer just before exposure, after 4 hours, and after 8 hours of treatment. No temperature rises or falls were observed.

When any rat from either group started to develop neurological signs of tumor growth, that rat and its match were euthanized by perfusion-fixation of the brains under chloralhydrate anesthesia. Five coronal slices of each brain were studied microscopically in cresyl violet staining, thereby covering the entire encephalon except the frontal and occipital poles. A pathologist who had no prior knowledge of the treatment of each rat examined all of the brains. The longest and shortest dimensions of each tumor in the slice where the tumor was most extended were measured, and the area of the tumor was calculated using the formula for the area of an ellipse.

All of the rats developed rounded, polycyclic tumors, with well-defined boundaries. Histopathologic examination showed the tumors usually to be solid with minor necrotic areas not correlated with treatment, tumor size, or time elapse from inoculation to death. There were no signs of brain damage outside of the tumor areas, either necrosis, gliosis, or inflammatory changes ascribable to RFEMF exposure.

The measurements of tumor area were expressed in terms of the tumor area of each RFEMF-exposed rat (E) and that of its matched control (C). The ratios E/(C-1) for each pair were averaged over the matched pairs for each form of RFEMF used, and were displayed in Table 2 of the paper as relative changes (in percent). Almost all of the relative changes well exceeded 100%; but from using Student's t-test on paired values, the authors obtained and displayed t and p values that were all nonsignificant. However, they gave no standard deviations. Instead, they stated: "The standard deviation is large, but this is the result of the great individual variation in the model where the status of the inoculated cells as well as that of the recipient animal is highly influential". They concluded that they had found no significant differences between the RFEMF-exposed and the control rats. Nevertheless, in their discussion they remarked that they had found some RFEMF-exposed rats with much larger tumors than their controls.

In view of the very large variabilities in the data for both the RFEMF-exposed and their matched control rats, the results of this study provide no credible evidence that the RFEMF did or did not alter tumor growth initiated by injection of RG2 cells into the brains of the rats.

In one of several studies, Lai and Singh (1995) sought to determine whether acute exposure of rats to 2.45-GHz RFEMF would affect the DNA in brain cells. A multiple waveguide system previously developed by Guy et al. (1979) for exposing small animals was used. The system consisted of eight circular waveguides excited by a single microwave source via a power splitter, to concurrently excite all of the waveguides in the circularly-polarized TE11 mode. The waveguides were constructed of galvanized wire screen, and within each was a plastic chamber for housing a rat.

In the first experiment, rats were exposed within each waveguide to 2-μs pulses of 2.45-GHz RFEMF, 500 pulses per second, for 2 hours at 1 or 2 mW/cm^2 spatially averaged power density. Those levels were said to yield average whole-body SARs of 0.6 or 1.2 W/kg based on dosimetric data of Chou et al. (1984). The authors, citing Chou et al. (1985), noted that at 0.6 W/kg, the local SAR in eight brain regions varied from 0.5 to 2.0 W/kg. Right after exposure or 4 hours later, the rats were euthanized and their hippocampi and the rest of their brains were assayed separately for single-strand DNA breaks. In the second experiment, rats were exposed for 2 hours to 2.45-GHz CW at 2 mW/cm^2 (1.2 W/kg whole-body SAR) instead of pulsed RFEMF, euthanized immediately or 4 hours post-exposure, and their whole brains were assayed.

In both experiments, rats were sham-exposed as controls. The assays were performed (without knowledge of which treatment each rat had received) by the method of Singh et al. (1994a), previously used to assess the damage to the DNA in human lymphocytes exposed *in vitro* to ionizing radiation. In the present study, preparations of DNA in brain cells of rats were assayed after exposure *in vivo* to the RFEMF. The method consisted of preparation of brain samples as microgels on slides for DNA unwinding and electrophoresis, with the latter technique involving use of an electric field to spatially spread any DNA fragments along such slides by their molecular weights.

At either assay time, the rats were placed in a closed box containing dry ice for 60 seconds and then decapitated, at which time the brains were removed, and the hippocampus of each was excised and assayed for DNA single-strand breaks, as was the rest of the brain. Right after dissection, the brain tissues were immersed in ice-cold phosphate-buffered saline (PBS), washed four times with the same buffer to remove most of the red blood cells, and minced in a centrifuge tube containing ice-cold PBS to obtain pieces of about 1 mm^3. Four more washings in the buffer removed most of the remaining red blood cells. The tissue pieces in 5 ml of PBS were then dispersed into single-cell suspensions, consisting of different types of brain cells.

A 10-μl sample of cell suspension was mixed with 0.3 ml of 0.5% agarose held at 37°C, and 75 μl of the mixture were pipetted onto a frosted slide and topped with a coverglass to make a microgel on the slide. The slide was then placed on ice for 1 minute to allow the cell/agarose to gel. The coverglass was removed, and 75 μl of agarose were layered on top of the cell/agarose.

After such preparation, slides were immersed in lysing solution at 0°C for 1 hour, and for detecting single-strand DNA breaks, they were treated with DNAase-free proteinase K in lysing solution that was preincubated for 1 hour at 37°C to ensure DNAase inactivation. Each slide was then placed on the horizontal slab of an electrophoretic assembly modified to permit connection of both ends of each electrode to the power supply, to render more uniform the electric field along the slide. An electrophoretic buffer was poured into the assembly to cover the slide to a height of 6 mm, and a period of 20 minutes was allowed for DNA unwinding.

Electrophoresis was done for 60 minutes (at 18 V and about 300 mA) with the buffer recirculating at 100 ml/min, at the end of which the extra buffer was removed from the top of each slide. At this point, the slides were processed in pairs three times for neutralization, dehydrated in absolute ethanol, and blow-dried, after which each slide was stained with a solution of YOYO-1 and topped with a slide cover. The

authors, citing Glazer and Rye (1992), noted that YOYO-1 is a dye that intercalates (is inserted) at various sites in DNA, and that it yields fluorescence about 500 times more intense than for more commonly used ethidium bromide.

Slides were analyzed with a Reichert Model 2071 fluorescence microscope (excitation at 490 nm, emission at 515 nm, and dichromic filter at 500 nm)., The electrophoretic movement of DNA fragments out of the cell nucleus was monitored for 50 representative cells on each slide, and the migration length, measured from the rim of the nuclear area to the last pixel of DNA at its leading edge, was used as the index of DNA single-strand breaks. The data were analyzed by ANOVA, and the differences between groups were compared by the Newman-Keuls test.

In the first experiment [using pulsed RFEMF], the mean migration lengths of DNA and the standard errors of the mean (SEMs) for the hippocampal cells were assayed immediately or four hours after whole-body exposure at 0.6 W/kg, 1.2 W/kg, or sham exposure. Those results were displayed as bar graphs in Figure 1 of the paper. The graphs were for eight rats that had been exposed at each RFEMF level for 2 hours, and 11 rats that had been sham-exposed. The numerical data were not tabulated, but the bar graphs for the immediate post-exposure assays showed that the mean DNA migration length for exposure at 0.6 W/kg and 1.6 W/kg were respectively larger and smaller than for sham exposure. The authors stated that by one-way ANOVA, there was no significant effect ($P>0.05$) of exposure at either RFEMF level.

The corresponding results for the assays four hours after treatment were reported as follows: By the Newman-Keuls test, the mean DNA migration lengths for exposure at either RFEMF level differed significantly ($P<0.01$) from that for sham exposure, but the difference between the values for the two exposure levels was not significant ($P>0.05$). For reasons not stated, the SEM bar for exposure at 0.6 W/kg was not displayed.

The results for the rest of the brains in the first experiment were similarly displayed in Figure 2 of the paper. The assays done right after treatment showed no significant differences among the exposures at either RFEMF level and sham exposure. However, those done four hours after treatment showed a larger mean DNA migration length for exposure at 0.6 W/kg than for sham exposure and a still larger mean DNA migration length for exposure at 1.2 W/kg than at 0.6 W/kg. As before, the SEM bar for exposure at 0.6 W/kg was absent.

For the second experiment (with 2.45-GHz CW RFEMF at 1.2 W/Kg only, sham exposure, and whole-brain assays), the results were presented as bar graphs in Figure 3 of the paper. Those for the assays immediately after treatment were for eight RFEMF-exposed rats but only seven sham-exposed rats, and those for the four-hour post-treatment assays were for eight each RFEMF-exposed and sham-exposed rats. The authors stated that the assays done at both times showed significant differences between RFEMF- and sham exposure in mean DNA migration length by both the two-way ANOVA ($P<0.005$) and the Newman-Keuls tests ($P<0.01$).

The authors also stated: "It should be noted that the baseline of DNA single-strand breaks in the sham-exposed samples in this experiment is lower than that in the previous experiment. This difference could be due to the use of whole brain in this study, whereas, in the previous study, the hippocampus was dissected out and assayed separately from the rest of the brain. The dissection process took 40-45 s more before the tissues could be put into ice-cold buffer for processing. Such a delay and the additional disturbance to the tissue during dissection could increase the baseline level of DNA breaks." [From the bar graphs, the mean baseline DNA-migration lengths obtained in the two experiments were respectively about 160 microns and 100 microns.]

The authors remarked that the interaction mechanism between RFEMF and DNA is unknown, and suggested that an increase in DNA single-strand breaks could be due to an RFEMF-induced increase in the rate of breaking or reduction in the DNA damage-repair processes in the cell. Apparently, possible effects on the damage-repair processes were the basis for the four-hour post-treatment assays.

Several aspects of this study are open to question. First and probably most important, no dosimetry measurements were performed; instead, the authors cited Chou et al. (1985) in which the local SARs in eight brain regions were said to differ widely at a specific whole-body SAR. Thus, quite uncertain are the actual SARs in the hippocampus, or in the rest of the brain in the first experiment and in the whole brain in the second experiment. Also unknown are any possible temporal variations of local SARs due to rat movements or changes in body orientation or configuration during the two-hour exposures.

Second, the small numbers of rats stated in the bar graphs seem to imply that only one group of rats was treated at each level in each experiment, with immediate and four-hour delays in the assays. If this were the case, then the reported positive findings, if real, were not statistically robust. Moreover, since 50 cells on each slide were selected for analysis, to what extent did the other cells vary from those selected as representative?

Third, in a study involving exposure *in vivo*, followed by tissue-sample extraction, processing, and assaying that all required some time to perform, tissue autolysis may have been an important uncontrolled factor.

Fourth, not clear is why the numbers of rats treated were different in each experiment. (Eleven rats were sham-exposed and eight rats were exposed at each of the two RFEMF levels in the first experiment, whereas only seven rats were sham-exposed and eight rats were exposed to the single RFEMF level in the second experiment). The question here is: were other rats treated but not included in the results presented, and if so, why?

Fifth, also not clear is whether the RFEMF exposures and sham exposures were performed concurrently. If not, what (if any) uncontrolled differences in ancillary conditions occurred between the two forms of exposure?

Thus, little if any scientific credence can be given to the findings of this study, primarily because of the large spatial and temporal uncertainties of the SARs in the brain, the unknown role (if any) of uncontrolled factors such as tissue autolysis, and the small numbers of rats indicated in the results.

Williams (1996) commented on the Lai and Singh (1995) study. First, the reported absence [in the first experiment] of an increase of single-strand DNA breaks (SSDB) assayed right after RFEMF exposure and a significant increase of SSDB assayed four-hours post-exposure, is not consistent with SSDB findings induced by other types of radiation. In the latter, the breakage revealed by alkaline denaturization is generally maximal at exposure end or shortly afterward, after which repair begins to ameliorate the damage, a point applicable to the rodent brain [citing three references]. Second, Williams (1996) also noted that the SSDB background [from sham exposure] in the second experiment was about 2/3 of the background in the first experiment, indicating significant artifact in the assay processes. Third, although the 20% migration increase found immediately after exposure to the CW RFEMF [in the first experiment] is consistent with findings with other types of radiation, the finding of no migration decrease assayed four hours after exposure to the pulsed RFEMF could be indicative of the absence of repair of the putative DNA breaks, a finding that is not consistent with the knowledge about processing of DNA damage resulting from other forms of radiation.

Lai and Singh (1996b), in response to Williams (1996), cited other references to indicate that the extent and the duration of the DNA repair processes after damage is not constant, but depends on the specifics of the experiments, such as the type of radiation, the exposure duration, and the details of the assay processes.

Lai and Singh (1996a) performed a similar study with rats, but toward seeking double-strand as well as single-strand DNA breaks in rat brain cells from exposure to the same pulsed and CW RFEMF for two hours. Only one RFEMF level was used: 2 mW/cm^2 average power density, said again to yield a mean whole-body SAR of 1.2 W/kg for either pulsed or CW RFEMF. Explicitly noted was that included in each session were both RFEMF-exposed and sham-exposed rats, and that separate groups of rats were sham-exposed for the pulsed and CW exposures. However, unlike in the previous study, the removal of

the brains and the assays were done only four hours after treatment. Thus, four groups of eight rats each were treated: one each with the pulsed and CW RFEMF and one sham-exposed group for each RFEMF group.

The processing of the excised brains was similar to that used in the previous study, to yield slides for alkaline microgel electrophoresis by the method of Singh et al. (1994a). The slides were processed to reveal DNA single-strand breaks (by treatment with DNAase-free proteinase K at 37°C for two hours, as in the previous study) and for double-strand breaks by treatment with ribonuclease-A for two hours followed by proteinase-K for two hours, both at 37°C. The cell preparations were stained with YOYO-1 and electrophoretic procedures used were similar to those in the previous study.

Four slides were prepared from a brain sample of each rat, two slides each for single-strand and double-strand breaks. Fifty representative cells were scored for each slide, i.e., 100 cells for single-strand breaks and 100 cells for double-strand breaks from each rat. The data on mean single-strand and double-strand migration lengths for the cells were compared with those from the corresponding sham-exposed rats with Student's two-tailed t-test, with $P<0.05$ taken as significant. In addition, the mean percentages of cells versus DNA migration length averaged over the eight rats of each group were plotted as bars in 10-μm intervals from 90-99 microns to 240-249 microns, and the differences in distributions between the respective RFEMF groups and their corresponding sham groups were compared by the chi^2 test.

Shown in Figure 1 of the paper were bar graphs of the mean length of DNA migration (and SEM) representing the single-strand breaks ascribed to exposure to the pulsed-RFEMF and the corresponding sham exposure, and similarly for the CW RFEMF. The mean migration lengths for both forms of RFEMF significantly exceeded ($P<0.01$) their respective lengths for sham exposure, but the mean lengths for the pulsed and CW RFEMF did not differ significantly ($P>0.05$) from one another. For exposure to the pulsed RFEMF, the plots of mean percentages of cells in the succession of 10-μm intervals, shown in Figure 2 of the paper, indicated far larger values of single-strand DNA breaks in the intervals 180-189 through 220-229 μm than for the corresponding sham exposure. However, the results for the intervals from 110-119 through 150-159 μm were opposite: far smaller percentages for the pulsed-RFEMF exposure than for sham exposure. The results for single-strand breaks from the CW exposure, shown in Figure 3 of the paper, were similar but less pronounced.

Shown in Figures 4-6 of the paper were the results for double-strand DNA breaks corresponding to those in Figures 1-3 for single-strand DNA breaks. The mean-migration-length bar graphs were similar to those for single-strand breaks, but also were less pronounced. However, the distributions for both the pulsed- and CW exposures were considerably different from those for the single-strand breaks. For both forms of RFEMF, the largest percentages of cells were found more centrally between the intervals 100-109 μm through about 190-199 μm, and the percentages for the corresponding sham exposures in that range of intervals in a number of cases were comparable to their RFEMF values.

As in the Lai and Singh (1995) paper, the lack of meaningful dosimetry and the unknown time variations thereof due to rat movements, changes in orientation relative to the direction of the incident fields, and changes in body configurations are the major criticisms. Also relevant is the possible confounding of the results from autolysis, a point that may not be applicable to the lymphocyte study *in vitro* cited in Singh et al. (1994a).

Lai and Singh (1997) hypothesized that free radicals were involved in the rat-brain DNA damages from exposure to the pulsed 2.45-GHz RFEMF *in vivo*, found in their previous studies, when the brain-cell assays were done four hours post-exposure. Accordingly, rats were exposed to the same pulsed RFEMF for two hours but were injected immediately before and after such exposure with melatonin or spin-trap compound N-tert-butyl--phenylnitrone (PBN), both said to be efficient free-radical scavengers. The expectation was that those substances would block the damage from the RFEMF.

Groups of rats were injected subcutaneously with 1 mg/kg by body weight of melatonin at a concentration of 1 mg/ml in a 1% ethanol-saline solution (the melatonin "vehicle"), or injected

intraperitoneally with 100 mg/kg of PBN at 25 mg/ml in physiological saline (the PBN "vehicle"). Those doses were both based on citations in the literature. For comparisons, other groups were injected with the same volumes of their respective vehicles only. The authors noted that because both drugs have a short half-life in the blood (0.5-2 hours) and that the exact time when DNA strand breaks occur was not known, they had decided to inject the rats both just before and right after exposure. Thus, four treatment groups for each drug were studied: RFEMF-drug [in its vehicle], RFEMF-vehicle [only], sham-drug [in its vehicle], and sham-vehicle [only]. In addition, a group of unhandled rats was included in each experiment, housed in their home cages during the entire time.

The assays for DNA strand breaks were done essentially by the method described in Lai and Singh (1996a), with the DNA migration length as the index for strand breaks. Two slides were prepared from the brain sample of each rat, one each for the assay of single-strand and double-strand breaks. Fifty cells were randomly chosen on each slide for scoring. However, extensively damaged cells, with DNA totally migrated out from the nuclear region, were not measured. The authors noted that such cells probably resulted from the tissue and cell-processing procedures, and that they occurred equally in RFEMF-exposed, sham-exposed, and unhandled samples. As before, the results were shown as bar graphs of mean migration lengths (and SEMs), and as distributions of the percentages of cells versus migration length in 10-μm bins.

Displayed in Figure 1 of the paper were the effects of treatment with melatonin on single-strand breaks. The mean migration length (MML) for the sham-vehicle group (7 rats) was approximately the same as for the untreated cage controls (9 rats): about 140 microns. The MML for the RFEMF-vehicle group (8 rats) was about 190 microns, significantly higher than for the sham-vehicle group, taken as an indication that the RFEMF had increased the MML. By contrast, the MMLs for the sham-melatonin and the RFEMF-melatonin groups (9 rats each) were about equal and only slightly higher than for the sham-vehicle group, results interpreted by the authors as confirming their hypothesis that the melatonin had blocked the effect of the RFEMF by scavenging free radicals.

Figure 2 of the paper showed the corresponding distributions of the cell percentages within the 10-μm bins. The results were less clear. The cage-control group had a split distribution of bins with non-zero cell percentages: one set in the range from 110 to 159 μm with a peak of about 32% of the cells in the 120-129 bin, and a smaller set in the higher range from 170 to 199 μm, with a peak of about 5% and a total of roughly 12% of the cells. For the sham-vehicle group, there was a single narrower range of bins with non-zero cell percentages from 110-119 μm through 150-159 μm, with peaks of about 27% each in the bins 130-139 μm and 140-149 μm. Presumably those differences in distribution were not inconsistent with the equal MMLs noted above for those two groups. However, the distribution for the sham-melatonin group covered a wider range than for the cage-control or sham-vehicle group: 119-219 μm, probably consistent with the slightly higher MML for that group.

The cell distribution for the RFEMF-vehicle group appeared to be more symmetric and it covered a higher range than any of the other groups: from 130 to 259 μm, indicating a larger MML than for the sham-vehicle group, taken as an effect of the RFEMF in the absence of melatonin. The range for the RFEMF-melatonin group was 110 to 199 μm, indicating a lower MML than for the RFEMF-vehicle group. Noteworthy was a closer overall resemblance between the distributions for the RFEMF-melatonin and the sham-melatonin groups than to the other distributions, a result taken as before as blockage of the RFEMF effect by the melatonin.

The double-strand MMLs with and without melatonin injection, displayed in Figure 3 of the paper, were similar to the corresponding single-strand MMLs. However, the distributions of the cell percentages between the sham-vehicle and the unhandled-control groups (Figure 4) showed larger differences than those for the single-strand groups, as did the differences between the RFEMF-vehicle group and those two groups. Again, however, the shape of the distribution for the RFEMF-melatonin group was similar to that for the sham-melatonin group. The results were less clear. The cage-control group had a split distribution of bins with non-zero cell percentages: one set in the range from 110 to 159 μm, with a peak of about 32% of the cells in the 120-129 bin, and a smaller set in the higher range from 170 to 199 μm,

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The results with PBN, shown in Figures 5 and 6 of the paper for single-strand breaks and in Figures 7 and 8 for double-strand breaks, were similar to those with melatonin except that all of the cell-distribution plots appeared to cover narrower ranges and smaller MMLs than the corresponding plots for melatonin.

Several points about this study are considered below.

One aspect of the experimental procedure was to inject rats twice with melatonin, i.e. just before and just after the two hours of RFEMF- or sham exposure, presumably to scavenge the free-radicals present before such treatment and those present immediately after treatment. The authors, citing several references, noted that melatonin can readily pass through the blood-brain barrier and the cell and nuclear membranes. After the first injection, how did those rats behave during those two hours, i.e. did they tend to sleep or otherwise differ in their behavior and/or metabolism from those not treated with melatonin? If there were such effects, perhaps they would have been manifested as significant differences in MMLs between the sham-melatonin and sham-vehicle groups.

As indicated previously, the authors mentioned that extensively damaged cells were not measured, that such cells probably resulted from the tissue and cell-processing procedures, and that they occurred equally in RFEMF-exposed, sham-exposed, and unhandled samples. Unclear is to what extent, if any, such damage had occurred on the slides assayed and whether such damage, if present, had influenced the selection of the cells thereon for measurement. Perhaps related were a few differences in the numbers of rats indicated in Figures 1, 3, 5, and 7, and the absence of standard-error-of-the-mean [SEM] bars for: the sham-vehicle group in Figure 1, the sham-vehicle and sham-melatonin groups in Figure 3, all groups except the RFEMF-PBN group in Figure 5, and all groups in Figure 7.

Most important, as in the earlier Lai and Singh papers discussed above, the lack of meaningful dosimetry and the unknown time variations thereof due to rat movements, changes in orientation relative to the direction of the incident fields, and changes in body configurations are the major criticisms. In addition is the possible confounding of the results from autolysis. Again, little if any scientific credence can be given to the findings of this study.

Wu et al. (1994) investigated whether colon cancer induced in young mice by dimethylhydrazine (DMH) would be affected by exposure to 2.45-GHz RFEMF (MW). For comparison, the effects of combining DMH with 12-O-tetradecanoylphorbal-13-acetate (TPA) were also studied.

The study was based on the belief that cells become cancerous in two successive stages: initiation by a physical or chemical agent that causes irreversible damage to the DNA in specific genes, and promotion by another agent that stimulates tumor growth by interfering with processes that regulate cell growth. In this study, the initiator used was DMH and the promoter was TPA. Accordingly, Balb/c mice of ages 4-5 weeks in about equal numbers of males and females were randomly divided into four groups:

Group A: controls (29 mice)
Group B: DMH only (28 mice)
Group C: DMH + MW (26 mice)
Group D: DMH + TPA (32 mice)

The mice to be exposed to the RFEMF were housed singly in Plexiglas cages of inner dimensions 8.5 x 3.5 x 3.5 cm, which allowed each mouse to reverse direction but generally forced it to maintain its long body axis parallel to the longest dimension of the cage. Mice were exposed dorsally with body axis parallel to the electric field in a 2x2 anechoic chamber at locations of power density 10 mW/cm^2, as measured with a Narda model 8616 broadband isotropic radiation monitor. From Durney et al. (1978), the SAR was estimated as 10-12 W/kg.

Exposures were for 3 hours daily, 6 days a week, for 5 months. The cage positions within the chamber were shifted counterclockwise after each exposure to obtain uniform total exposure for each mouse. The chamber temperature was maintained at about 25 ± 1°C in summer and 22 ± 1°C in winter. The humidity was held at 50% ± 5% during January-March and 76% ± 5% during April-June. Not stated was the kind of antenna used within the chamber or how many sites therein showed approximately the stated power density. Sham-exposed mice were treated similarly but with no RFEMF. From the description, however, apparently the RFEMF- and sham exposures were not done concurrently.

DMH, an initiator, was dissolved in 0.9% saline to a concentration of 0.15% or 0.2% just before use. Once per week for 14 weeks, the mice of Groups B, C, and D were injected subcutaneously with the 0.15% solution at a dose of 0.1 ml per 10 g of body weight (15 mg/kg). For the next 8 weeks, they were injected with the 0.2% solution at 0.1 ml per 10 g (20 mg/kg). The mice in Group A (controls) were injected with 0.9% saline at 0.1 ml per 10 g instead of DMH.

Five milligrams of TPA, a promoter, was dissolved in 50 ml of acetone, separated into ampules containing amounts needed for each injection. The quantity in each ampule was vacuum-dried, and the ampules were sealed and frozen. Just before use, the dried TPA was dissolved in 0.9% saline to a concentration of 0.001%, and 0.2 ml of the solution was injected i.p. once a week in the mice of Group D (2 μg/mouse)for 10 weeks starting 3 weeks after the initial treatment with DMH. Those in Groups A, B, and C were injected with 0.2 ml of 0.9% saline instead of TPA.

At week 25, all mice were euthanized by cervical dislocation. The colon was removed and slit lengthwise along the mesentery side, the intestine was cleaned, and the mucose was stained with 1% methanol blue. Nodules in mucosa were observed with an anatomical scope. All intestines and parts of nodules cut from mucosa were fixed in formalin, mounted on slides, stained with hemotoxylin-eosin, and observed by light-microscopy.

The authors observed that during the experiment, nodules exceeding 3 mm in diameter on mucosa were easily distinguishable as tumor (cancer) nodules or as lymph nodules by their differences in shape and surface appearance; for nodules smaller than 3 mm, however, this distinction could not be made. Thus, diagnoses of those larger than 3 mm were done by anatomical scope, and those smaller than 3 mm were done by microscopy.

Tumor incidences in the four groups are shown in Table 33 (adapted from Table 2 of the paper).

TABLE 33: COLON TUMOR INCIDENCE AMONG MOUSE GROUPS
[Wu et al. (1994)]

Group Incidence	No. of Mice	No. of Mice with Tumors	Tumor Incidence (Percent)
A (controls)	29	0	0
B (DMH)	28	13	46.0
C (DMH + MW)	26	13	50.0
D (DMH + TPA)	32	17	53.0

It is seen that none of the control mice developed colon tumors. The authors noted that by chi^2 test, the differences among Groups B, C, and D were not significant. Thus, evidently DMH functioned as an initiator in those three groups, but RFEMF exposure did not augment the effect of DMH (Group C) nor did TPA (group D).

Displayed in Figure 1 of the paper were bar graphs of the mean numbers of tumors per tumor-bearing mouse for Groups B, C, and D. Those results respectively were 2.76 ± 0.4 (SD), 3.38 ± 0.49, and 7.18 ± 1.25. The mean for Group D was significantly higher than for Groups B and C ($P<0.05$), but the difference between Groups B and C was nonsignificant ($P>0.05$).

Also shown in Figure 1 were the median percentages of mice with more than 3 tumors each and the median percentages with lesions larger than 5 mm in tumor-bearing mice. The results for the former endpoint were 23%, 30%, and 58.9% respectively in Groups B, C, and D; by chi^2 test, the value for Group D was significantly higher than for Groups B and C, and the difference between the latter two values was not significant ($P>0.25$). The results on lesions larger than 5 mm in tumor-bearing mice were: 30.8% for Groups B and C, and 70.6% for Group D, with the latter significantly higher than for B and C.

In addition, the percentages of protuberant and invasive foci were 13.9% in Group B, 16.3% in Group C, and 44.1% in Group D, with the latter group also significantly higher than for B and C, and with the difference between B and C not significant.

All of the results above indicate that prolonged exposure of young mice to 2.45-GHz at 10 mW/cm^2 (estimated SAR 10-12 W/kg) had no effect on tumor incidence or growth caused by injection of the initiator DMH alone or in combination with the promoter TPA. As noted previously, the antenna and hence the exposure arrangement were not adequately described. Also unclear was how the specific numbers of mice in each group (Table 33) were selected. Such points tend to reduce confidence in the negative findings but not fatally so.

Repacholi et al. (1997) used a specific strain of transgenic mice to determine whether exposure for 30 minutes twice daily over a period of about 18 months to RFEMF at a frequency used in digital mobile telecommunications in Australia would increase the spontaneous incidence of lymphoma of that strain. [The adjective "transgenic" generally refers to organisms having chromosomes that contain copies of genes derived from other species or strains.] For this study, transgenic mice having the Eμ-*Pim1* gene (which renders them prone to the spontaneous development of lymphoma), were successively crossbred with nontransgenic mice to obtain offspring 5-10% of which would carry the Eμ-*Pim1* gene and would develop lymphoma during the first 10 months of life (with 25% developing the disease during their lifetimes). The mice were bred under specific-pathogen-free (SPF) conditions in California and air-freighted at age about 2 months to an SPF research facility in Australia.

The authors exposed 101 virgin female mice to 0.6-millisecond, GSM-modulated 900-MHz RFEMF at 217 pulses per second for two 30-minute periods a day for up to 18 months, starting at age 2 months. ["GSM "stands for Global System for Mobile Communication.] As controls, 100 mice were similarly sham-exposed. The SPF status of the animals was maintained for the duration of the study.

For RFEMF exposure, the mice were housed in a small room (2.6 m by 2.2 m by 2.45 m), the inner walls of which were lined with aluminum sheeting as shielding to avoid interfering with the Australian mobile phone system. A vertical ground plane was mounted parallel to one wall, and a horizontal quarter-wave monopole antenna was mounted at its center. Affixed to the ground plane in a circular array were 20 15-cm by 30-cm Lucite stands, the centers of which were 65 cm from the monopole, and a polycarbonate cage holding 5 mice was placed on each of the stands. Sawdust bedding, food pellets, and a glass bottle of water end-mounted relative to the incident RFEMF (to minimize perturbations) were provided for each cage. Twice each week, the cages were cleaned, and fresh pellets and water were provided. The sham exposures were done in an adjacent, slightly narrower room (2.6 m by 1.8 m by 2.45 m) that had a ground plane and an unenergized monopole.

The RFEMF levels at each cage position were measured using calibrated commercial instruments, while the other 19 fully equipped cages of mice were present. The power densities ranged from 2.6 to 13 W/m^2 (0.26 to 1.3 mW/cm^2). The authors remarked: "Numerous measurements of the field distribution inside the room were conducted to assess the interference patterns produced by reflections from the aluminum walls. While significant variations could be detected in the room, the variation in the vicinity of the animal cages was within the range of values given above."

SARs were determined at 900 MHz in three tissue-equivalent phantoms as representations of a small, medium, and large mouse. The measurements were performed within a "shielded semi-anechoic room", with each phantom placed 70 cm from the central axis of the open face of a WG-4 waveguide flange serving as the antenna. The internal dimensions of the flange face were 12.4-cm x 24.8-cm, and the phantom site was said to be in its far field. The phantom shapes were derived from outline traces of mice weighing 26, 34, and 64 g.

The internal electric field of each phantom mouse was measured with a probe inserted at three points along its long body axis, and with each phantom in three orientations relative to the incident RFEMF direction. The SARs that were determined from measurements of the electric field at the three points of each phantom were averaged. Those averages ranged from a low of 0.0078 W/kg for the small phantom mouse exposed at 2.6 W/m^2 (0.26 mW/cm^2) with its axis perpendicular to the electric field vector (H-polarization), to a high of 4.2 W/kg for the medium phantom mouse exposed at 13 W/m^2 (1.3 mW/cm^2) with its axis parallel to the electric field (E-polarization). These values correspond to a possible 500:1 range in exposure level of the experimental animals. The normalized SARs were a low of 0.003 W/kg per W/m^2 and a high of 0.31 W/kg per W/m^2. However, such averages do not appear to represent whole-body SARs as usually defined.

The primary results of the study are displayed in Table 34 (adapted from Table III of the paper) in terms of the incidences of: lymphoma, characterized as lymphoblastic [LB] or non-lymphoblastic [Non-LB]; renal disease (terminal glomerulopathy) only [REN] or with both lymphoma and renal disease [REN+LYM]; other diseases [OTHER] (deaths from miscellaneous causes, including dehydration, injuries, hepatoma, and amyloidosis); and undiagnosable cases [UNDIAG] (dead mice with tissues too autolysed for analysis).

TABLE 34: LYMPHOMA AND OTHER DISEASES AMONG RFEMF-EXPOSED TRANSGENIC MICE
[Repacholi et al. (1997)]

GROUP	LB	NON-LB	TOTAL	REN	REN+LYM	OTHER	UNDIAG
Control (100)	3	19	22	7	11	8	7
Exposed (101)	6	37	43	8	10	12	7

Up to about age 10 months, thymic LB lymphoma was predominant in both the exposed and control groups of transgenic mice, a finding the authors expected from [non-RFEMF] studies of *Pim1* mice cited in the literature [van Lohuizen et al. (1989) and Breuer et al. (1989)]. However, the authors stated that the disease was recognizable only at a stage when the mice had begun to suffer respiratory stress, and that the first three cases were found dead in the cage but could be diagnosed by the histological

appearance of the tissues. As the table shows, 6 exposed mice and 3 control mice were diagnosed with LB lymphoma. At ages older than 10 months, cases of non-LB lymphoma began to appear, totaling 37 exposed mice and 19 control mice by the age of 18 months (experiment end). Histologically, the disease was manifested in various forms (mostly follicular lymphoma in the spleen), but none were lymphoblastic.

The authors indicated that by the conditional binomial exact test, the difference between the totals of 43 lymphoma cases of any type in the exposure group and the 22 sham-exposed cases was significant ($P<0.001$). In addition, by multivariate analysis using logistic regression, in which adjustments were made for age and body-weight differences and for mice that died from any type of lymphoma, the authors reported an odds ratio of 2.42 with a 95% confidence interval of 1.3-4.5 ($P<0.006$). Similarly, the difference in incidence between exposed and control groups for LB lymphoma (6 exposed versus 3 control cases) was nonsignificant.

The renal disease shown in the table initially appeared at 5-8 months of age in a few terminally ill mice. It reached a cumulative incidence of about 10% in both the control and exposed groups at age 18 months (at completion of the experiment), and was the sole cause of terminal illness in 7-8% of the mice. It also appeared in variably milder form in mice killed with other predominant diseases. The authors ascribed the renal disease to a product of transgene action unrelated to RFEMF exposure. Thus, its appearance in this study may indicate that the transgenic mice were already compromised with respect to possible effects of RFEMF.

The 12 exposed and eight control mice with "other disease" had exhibited abnormal clinical signs, but the authors found no evidence of lymphoma when the mice were killed. In seven cases of each group, no cause for illness could be discerned because their tissues were too autolysed. The mice with all diseases comprised 71% of the RFEMF-exposed group and 48% of the sham-exposed group, so 29% and 52% of the respective groups presumably were not affected.

In Figure 4 of the paper, the cumulative probabilities of LB- and non-LB lymphoma development were plotted versus age in months. The values were based on adjustments for losses of mice from causes other than lymphoma. However, few details of the calculations were given, particularly about the ages at illness detection or death of the individual mice or the numbers of mice that corresponded to each cumulative-probability point plotted. Based on coarse scaling of the coordinates of the graphs, apparently the first appearance of LB lymphoma had occurred at age 4 months (2 months after treatment start). At that time, the cumulative probability for LB lymphoma was roughly about 0.03 for both the exposed and control groups and remained about the same up to about age 10 months.

Between 10 and 11 months, the cumulative probability of the exposed group for LB lymphoma jumped from about 0.03 to about 0.1 and rose more gradually to about 0.17 by 19 months, whereas it remained at about 0.03 for the control group. Presumably the jump in probability for the exposed group was not significant because of the small total numbers of cases.

The graphs for non-LB lymphoma showed no cases in either group up to about age 10 months. Shortly afterward, the cumulative probability of the exposed group rose almost linearly to about 0.14 at 13 months, remained at that value to about 16 months, and rose again approximately linearly to about 0.6 at age 19 months. The cumulative probability of the control group also increased, but the rise started at about age 13 months and rose to only about 0.24 at age 19 months.

As discussed below, this study had serious flaws in its engineering and dosimetric aspects, interpretive problems with the use of transgenic mice as subjects, methodological problems with the protocol used for feeding and housing the mice and observing them during the 18-month period of exposing them twice daily for 30 minutes, the statistical treatment of the data, and problems with the bioeffects aspects per se about the ability to relate the findings to humans (non-transgenic).

Regarding the engineering and dosimetric aspects, the authors stated incorrectly that the mouse stands were located in the far field of the monopole. By lining the inside walls of the exposure chamber with aluminum, in essence they essentially created a reverberant microwave cavity having a multitude of

complex field modes, in which the term "far field" (propagation that is subject to the inverse-square law with no reflections) does not apply. Any mouse movements would vary such modes unpredictably and alter the amounts of energy absorbed by each mouse relative to the others, a problem compounded by housing the mice five to each cage and the consequent interactions among them in each cage. The large spread of the values (average power densities from 2.6 to 13 W/m^2; "normalized" [not whole-body] SARs ranging from 0.003 to 0.31 W/kg per W/m^2) for a possible total range of individual SARs of about 500:1 is severely problematical.

A basic difficulty was the smallness of the exposure chamber and the necessity for using metal shielding, apparently because of the unavailability of a much larger anechoic chamber lined internally with RF-absorbing material of sufficient sizes for use at 900 MHz. Such a chamber would have obviated the need for the shielding that impacted so negatively on the densitometry. Thus, little if any meaning can be given to those measurements and any conclusions about the role of RFEMF in the reported findings other than a statement that one group was exposed to RFEMF at unknown spatially and temporally varying individual levels and the "control" (sham-exposed) group was not exposed.

The use of transgenic mice of which only 5-10% would spontaneously develop lymphoma during the first 10 months of age seems inappropriate, especially because the sham-exposed mice were also susceptible to lymphoma and therefore already compromised. The unavailability of colony-control mice that were neither RFEMF- nor sham-exposed, for comparisons with the sham-exposed mice, is a basic problem with this strain of transgenic mice. Moreover, there is no evidence that the *Pim1* gene is present in humans.

The indicated practice of changing the sawdust bedding in the cages only twice weekly (when adding fresh food and water) could have resulted in ammonia toxicity from urine accumulation [which might have been the cause of the renal disease observed in both the RFEMF-exposed and sham-exposed mice]. Also, this possible toxicity problem might have been exacerbated by heating of the bedding from the higher levels of RFEMF. The authors indicated that the mice were inspected daily and were disturbed to check their mobility. Not clear was whether such inspections were done seven days a week, and if so, why severe autolysis could have occurred in some cases. Also not clear was whether the mice found dead had been replaced with live mice in their respective cages during the course of the experiment, to maintain the same exposure situation for the remaining mice.

To the credit of the authors, some of the points above were alluded to in their discussion as explanation, but also possibly as an expression of their uncertainty about the validity of the findings.

In sum, the data presented in Table III [Table 34 above] and Figure 4 of the paper, and the description of their statistical treatment were too sparse to permit an independent verification thereof. Possible verification may be moot in view of the basic flaws of the study. It should be noted that humans do not carry the *E-Pim1* gene and that the lymphomas that do occur in humans are categorized differently from those used by the authors for the mice. These reasons render it inappropriate to attempt to interpret the findings of this study as evidence for possibly analogous findings in humans. Thus, very little if any scientific credence or relevance can be ascribed to the findings herein about any relationship between RFEMF exposure and human health. Moreover, any endeavor to replicate this study by using the same experimental design and protocols would be fruitless.

Toler et al. (1997) investigated whether chronic exposure of mammary-tumor-prone mice to 435-MHz RFEMF at low levels would promote earlier onset, faster growth rate, or greater total incidence of mammary tumors. Two hundred female C3H/HeJ mice were exposed concurrently for 22 hours a day, 7 days a week, 21 months to horizontally polarized, pulsed 435-MHz RFEMF (1.0-μs pulses at 1-kHz pulse rate) at 1.0 mW/cm^2 average power density. The RFEMF values were selected to simulate exposure to the emissions from the U.S. Air Force's PAVE PAWS surveillance tracking system, which operates in the frequency band 420-450 MHz.

The authors noted that among the reasons for selecting C3H/HeJ mice for study were that they possess a milk-borne virus that induces spontaneous neoplasms in the mammary gland. The total

incidence and latency to tumor onset are well documented in colony-raised C3H/HeJ mice, and such tumor incidence is known to be altered by stressors or chemical agents such as confinement, dietary restriction, or melatonin administration. They also mentioned that this mouse strain was studied by Szmigielski et al. (1982).

The exposure system comprised four horizontal parallel-plate waveguides stacked vertically, with each waveguide consisting of two circular plates 3.66 meters in diameter and spaced 0.47 meters apart. A slotted-cylinder antenna was mounted at the center of each waveguide to yield horizontal polarization. Fifty mice housed individually in Plexiglas cages were positioned around the circumference of each waveguide. Each cage was fitted with a slotted Nalgene top consisting of a transparent polyvinyl chloride body that supported paper filter inserts. The mice were provided food and water ad libitum, with the food via a Plexiglas food hopper attached to each cage and the water from a bottle with a glass sipper tube perpendicular to the electric field. Two hundred other mice were similarly arranged and were sham-exposed in identical nonenergized waveguides, and 50 mice served as sentinels for health-status evaluations.

The SAR was determined by two methods. In the first method, each mouse was assumed to be ellipsoidal with major axis parallel to the electric field, and the calculated data in Durney et al. (1986) for 435 MHz was used, which yielded an SAR of about 0.1 W/kg per mW/cm^2. Calorimetry of RFEMF-exposed mouse carcasses was used in the second method. On the day before measurement of each mouse, it was euthanized, weighed, inserted into a prototype Plexiglas exposure cage, and placed on the waveguide perimeter overnight for thermal equilibration with the ambient temperature. A calorimeter filled with 125 g of water was concurrently equilibrated overnight. In the morning, the carcass was exposed to the RFEMF at 1.0 mW/cm^2 for 30 minutes, near the end of which the temperature of the water was adjusted to be 1-2°C below ambient. At end of exposure, the carcass was transferred into the calorimeter, the temperature was monitored until equilibrium was reached, and the resulting temperature rise was used to calculate the SAR of the carcass. Many such trials yielded an average SAR of 0.32 W/kg.

During a three-week acclimation period, 5 RFEMF-exposed and 5 sham-exposed sentinel mice were necropsied for pathology and quality assurance, which included acquisition of serum samples, intestinal cultures, direct fecal smears and perianal tape samples. Training in necropsy, palpation for tumors, and techniques of tissue preparation and fixation was conducted during this period.

One month after delivery of the mice, RFEMF exposure was started for the 200 mice in the RFEMF group and 20 in the sentinel group (presumably also the sham exposure of the 200 controls). On Monday through Thursday of each week during such treatment, 50 RFEMF-exposed and 50 sham-exposed were removed each day for inspection, palpation for tumors, and weighing, after which they were returned to clean cages with clean water bottles and sipper tubes. It was noted that throughout the experiment, the mice remained free of infectious diseases and parasites while housed in conventional rather that SPF (specific pathogen free) conditions.

Mice that were found dead during the treatment period were necropsied immediately if possible, and a tabulated list of 31 tissues were harvested and preserved in formalin. If immediate necropsy was impossible, the mice were refrigerated and necropsied within 24 hours. The same necropsy procedures were used for the mice euthanized because of the advanced state of their tumors (moribund sacrifices). Halothane inhalation was used for all sacrifices.

The evaluation of the health status and necropsy of 5 sentinel mice each at 6, 12, and 18 months of treatment were performed. At 21 months following treatment, exposure was terminated, the surviving mice (47 RFEMF-exposed and 47 sham-exposed) were euthanized and necropsied, and tissues therefrom were preserved and given detailed histological examination without knowledge of which mice had been RFEMF- or sham-exposed. The results were entered into a computerized data collection system (LABCAT).

Assessed for each group were the numbers of mice with neoplastic and non-neoplastic tumors or lesions at specific sites. The data were analyzed by logistic regression (using linear and quadratic terms), with tumor incidence modeled as a function of exposure and time. The tumor rates for the RFEMF-exposed and sham-exposed groups were compared using the likelihood-ratio score test on the regression coefficient. The use of logistic regression was based on the assumption that the diagnosed tumors were not directly responsible for the death of each mouse, and so the life-table test, appropriate for rapidly lethal tumors, was also applied. In addition, a chi^2 test based on the overall proportion of tumor-bearing mice was performed. Also estimated was the probability of survival and the differences in survival rates between the RFEMF-exposed and sham-exposed mice, using the product-limit procedure of Kaplan and Meier (1958).

The types and numbers of neoplasms in the most commonly affected organs, together with the chi^2 and probability values, were displayed in Table II of the paper. The major results therein are summarized in Table 35.

TABLE 35: NEOPLASMS OF THE MAMMARY GLAND, OVARY, LIVER, UTERUS, AND ADRENAL GLAND
[Toler et al. (1997)]

ORGAN	RFEMF-EXPOSED	SHAM-EXPOSED	CHI^2	P
Mammary Gland				
Number of Mice examined	193	190		
Adenocarcinoma	77	74	0.04	0.85
Adenocarcinoma, multiple	8	8	0.00	0.97
Total Adenocarcinoma	85	82	0.03	0.86
Ovary				
Number of Mice examined	177	166		
Epithelial stromal tumor	18	17	0.00	0.98
Bilateral epithelial stromal tumor	10	2	5.01	0.03
Total epithelial stromal and granulosa cell tumors	34	29	0.17	0.68
Liver				
Number of Mice examined	188	180		
Total hepatocellular adenoma	100	94	0.03	0.85
Total hepatocellular carcinoma	12	9	0.33	0.57
Uterus				
Number of Mice examined	187	172		
Total adenocarcinoma	16	18	0.38	0.54
Adrenal Gland				
Number of Mice examined	183	175		
Medulla: pheochromocytoma	9	6	0.49	0.48
Subcapsule: Adenoma	4	1	1.69	0.19

As indicated above, a total of 85 adenocarcinomas of the mammary gland appeared in 77 of 193 RFEMF-exposed mice versus 82 adenocarcinomas in 74 of 190 sham-exposed mice, incidences not significantly different between the groups. The authors noted that the morphology of the mammary carcinomas found was consistent with that expected in a high-incidence mouse strain such as one having the mammary tumor virus.

The incidence of mammary tumors found by palpation in both groups of mice and shown by necropsy to be adenocarcinomas was analyzed. The results were displayed as Kaplan-Meier plots of the

estimated probability of being tumor-free versus number of days of RFEMF exposure or sham exposure. The probability plots for both groups curved upward and were essentially parallel. The authors stated:

"A one-tailed continuity-corrected life-table analysis indicated that the differences observed in the plots could be reasonably attributed to chance variation in the times of tumor onset (P=0.317). Overall, the Kaplan-Meier estimated tumor probabilities, which adjust for differences in survival patterns, provide no evidence of an increased risk of mammary tumors due to exposure to RF radiation by the end of the study period (estimated rate for sham exposure = 55.7%, rate for exposure = 51.8%.)"

Also analyzed was the time of tumor onset in the subpopulations after the mice that never developed tumors were excluded. The median times for onset in the RFEMF-exposed and sham-exposed mice were respectively 470 and 479 days. By one-tailed Mann-Whitney U test, the difference was nonsignificant (P= 0.091). Plots of mammary-tumor growth rates, expressed as the cumulative mean spherical volume (measured with calipers) versus time for the period from palpation time to euthanasia (7-9 weeks), showed essentially no difference in growth rate between the groups.

Regarding tumor development in other organs or tissues (also displayed in Table 35), the only significant difference between the groups was for ovarian bilateral epithelial stromal tumor: chi^2=5.01, P=0.03. However, only 10 of 177 RFEMF-exposed mice and 2 of 166 sham-exposed mice were afflicted, and for the total numbers of mice with epithelial stromal tumors or granulosa cell tumors (34 RFEMF-exposed mice and 29 sham-exposed mice), chi^2 was 0.17 and P was 0.68 (nonsignificant).

Thus, no significant differences were found in the incidence of mammary tumors, latency to onset or growth of such tumors, or survival time of C3H/HeJ mice prone to such tumors from exposure to pulsed 435-MHz RFEMF at 1.0 mW/cm^2 average power density for 21 months. In addition, tumor development in other organs or tissues did not differ significantly between RFEMF-exposed and sham-exposed mice. As noted by the authors, their findings were consistent with those of Santini et al. (1988) with C57/6J mice, Wu et al. (1994) with BALB/c mice, and Frei et al. (1998) with C3H/HeJ mice. Also, they support the findings of Chou et al. (1992) with Sprague-Dawley rats. In addition, they noted that Szmigielski et al. (1982) had found that cancer-prone C3H/HeA mice exposed to 2.45-GHz RFEMF at 15 mW/cm^2 for 2 hours a day, 6 days a week for several months, developed tumors earlier than the sham-exposed controls. The latter authors suggested that the response might have been due to chronic stress.

Vijayalaxmi et al. (1997) exposed 100 mice of the C3H/HeJ strain prone to mammary tumors to 2.45-GHz CW RFEMF for 20 hours a day, 7 days a week, for 18 months at a whole-body SAR of 1.0 W/kg. The exposures were carried out in circularly polarized waveguides used in the University of Washington chronic study with rats [Guy et al. (1983); Chou et al. (1992)]. Each waveguide was modified to house two mice (instead of one rat), with free access to food and water. SAR was determined from waveguide measurements of forward, reflected, and absorbed power, and by a calorimetric method for small rodents [Padilla and Bixby (1987)]. One hundred C3H/HeJ mice were concurrently sham-exposed in such waveguides as controls. Twenty-five other C3H/HeJ mice were maintained as sentinel animals and used for periodic examination of health status. During the 18 months, the mice were visually inspected twice daily and were weighed, examined, and palpated for tumors weekly.

The objective of this study was to determine the extent of cytogenetic damage in peripheral blood and bone marrow cells from such RFEMF exposure, using the micronucleus assay. As noted by the authors, micronuclei arise from acentric chromosome fragments or whole chromosomes that are not incorporated into daughter nuclei during cell division. The micronucleus assay can detect both chromosome-breaking agents (clastogens) and agents that affect the cell spindle apparatus [microtubules in the cytoplasm that connect to the chromosomes during the metaphase of cell division (mitosis)], resulting in chromosome aberrations. The rodent micronucleus assay has been used widely for detecting genotoxic agents. In another study (not yet published to date), the primary objective was to determine whether such chronic RFEMF exposure of mammary-tumor-prone C3H/HeJ mice would promote the earlier onset, greater incidence, or faster growth rate of mammary tumors.

The survivors of the 18-month period were euthanized. They comprised 62 RFEMF-exposed, 58 sham-exposed, and 7 sentinel mice. Just before necropsy, a small drop of peripheral blood was collected from each mouse, placed on a clean microscope slide, and manipulated to produce a thin smear (about 2-3 cm across). At necropsy, marrow from one femur was flushed with 0.5 ml of fetal calf serum into a microfuge tube using a 1-ml syringe fitted with a 22-gauge needle. The cells were concentrated by gentle centrifugation, a small drop of resuspended cells was placed on a clean slide, and a smear was made. All smears were air-dried, fixed in absolute methanol, stained with buffered acridine orange, air-dried, and stored in black boxes.

The seven sentinel mice that survived the 18-month period were used as positive controls. They were injected with mitomycin C (MMC) at 1 mg/kg body weight, a clastogen known to induce micronuclei, and were euthanized 24 hours later. At that time, smears of peripheral blood and bone marrow were prepared and treated as described above.

Each of the slides was examined with a fluorescence microscope at 1000x magnification and blind-coded. Young polychromic erythrocytes (PCEs) were identified by their orange-red color, mature erythrocytes by their green color, and micronuclei by their yellowish color. The percentage of PCEs for each mouse was determined from examining 1,000 erythrocytes in peripheral blood and 200 erythrocytes in bone marrow. Also, 2,000 consecutive PCEs in the peripheral blood and bone marrow of each mouse were examined for the incidence of micronuclei.

Table 36 (adapted from Table I of the paper) displays the micronucleus frequencies in the peripheral blood and bone marrow of the RFEMF-exposed, sham-exposed, and MMC-treated mice.

TABLE 36: MICRONUCLEUS FREQUENCIES IN THE PERIPHERAL BLOOD AND BONE MARROW OF CANCER-PRONE MICE
[Vijayalaxmi et al. (1997)]

GROUP	NO. OF MICE	TOTAL ERYTHROCYTES	PERCENT PCEs PER 2,000 PCEs (±SD)	MICRONUCLEI PER 1,000 PCEs (±SD)
		PERIPHERAL BLOOD		
RFEMF-Exposed	62	62,000	2.8 (0.69)	4.5 (1.23)
Sham-Exposed	58	58,000	3.1 (0.71)	4.0 (1.12)
MMC-Injected	7	7,000	2.7 (0.68)	50.9 (6.18)
		BONE MARROW		
RFEMF-Exposed	62	12,400	51.5 (3.80)	6.1 (1.78)
Sham-Exposed	58	11,600	52.1 (3.32)	5.7 (1.60)
MMC-Injected	7	1,400	46.4 (3.59)	55.2 (4.65)

As originally reported: by Student's t-test, the differences in group mean percentages of PCEs in either peripheral blood or bone marrow among the RFEMF-exposed, sham-exposed, and MMC-treated mice were not significant. In addition, the numbers of micronuclei per 2,000 PCEs for the RFEMF-exposed and sham-exposed mice did not differ significantly, but the numbers for those treated with the clastogen MMC as a positive control were tenfold higher ($p<0.001$) in both peripheral blood and bone marrow than for the RFEMF-exposed and sham-exposed groups.

Mammary tumors were found in 12 of the 62 RFEMF-exposed mice and in 8 of the 58 sham-exposed mice. As shown in Table II of the paper, the group mean percentages of PCEs in the peripheral blood of the mice with tumors were 2.8 ± 0.71 and 3.2 ± 0.44 respectively for the RFEMF-exposed and sham-exposed mice. The values for bone marrow were 50.3 ± 0.73 and 50.2 ± 0.50. Both differences were nonsignificant.

However, in Vijayalaxmi et al. (1998a, 1998b), the authors noted that although the mean micronuclei in the last column of both tables were correctly given as per 1000 PCEs, the SDs displayed had been

calculated per 2000 instead of 1000 PCEs as stated. When they calculated the values correctly, use of the two-tailed Student's t-test indicated that the difference between the 62 RFEMF-exposed and 58 sham-exposed mice was statistically significant for both peripheral blood and bone marrow. However, the increase was only 1 micronucleus per 2000 PCEs (0.05%), a tiny change in a large number of mice exposed to the RFEMF over 18 months.

The authors indicated that their results demonstrate that the division and maturation of nucleated erythropoietic cells have not been inhibited by chronic exposure of tumor-prone C3H/HeJ mice to 2.45-GHz RFEMF. They cited the review by Salamone and Mavournin (1994) on spontaneous frequencies of micronucleated PCEs in bone marrow of more than 55 different mouse stocks used as untreated, sham-treated, or solvent-treated controls in various investigations, and noted that the values in their study were similar to those in that review. They concluded that such chronic exposure to 2.45-GHz RFEMF (the frequency that approximates the highest energy absorption rate [resonant frequency for maximum whole-body SAR] in mice) did not show any genotoxicity.

The experimental protocol and dosimetry used in this study are highly credible, and the finding of no genotoxicity is reinforced by the positive results obtained with the mice injected with mitomycin C, a known chromosome-breaking agent (clastogen).

Frei et al. (1998) investigated whether chronic, low-level exposure of mammary-tumor-prone mice to RFEMF promotes earlier onset, greater total incidence, or faster growth rate of mammary tumors. They exposed 100 C3H/HeJ mice to 2.45-GHz CW RFEMF in circularly polarized waveguides for 20 hours a day, 7 days a week, for 18 months at a whole-body SAR of 0.3 W/kg, and sham-exposed 100 other mice similarly.

The exposure system consisted of 100 independent cylindrical waveguides that could be excited by circularly polarized RFEMF, of the kind described by Guy et al. (1979) that were shown to provide relatively constant field coupling to each animal regardless of its position, changes of body configuration, or movements. RFEMF- and sham exposures were done concurrently, with the roles of the waveguides alternated periodically. The system was housed in two rooms at the Armstrong Laboratory, Brooks Air Force Base, Texas. It was a version of the system used in the chronic study of rats at the University of Washington [Chou et al (1995)] in which the Plexiglas cage within each waveguide was partitioned to hold two mice each (instead of a single rat), provision for free access to food and water by the mice, and easy removal of feces and urine.

Dosimetric measurements were done by calorimetry with mouse carcasses in various locations, and by computerized acquisition and storage of the forward, reflected, and absorbed RFEMF powers, the latter accounting for the specific powers absorbed by the cages and mice. Calorimetry of single carcasses placed midline and centered in cages, with long body axis parallel to the central line of the waveguide yielded a mean normalized SAR of 8.4 ± 2.8 (SD) W/kg per input watt. For two mice in various locations per cage, the mean normalized SAR was 6.4 ± 1.2 W/kg per watt; from the differential power-absorption measurements it was 6.9 ± 1.4 W/kg per watt, in reasonable agreement. The mean input power was set at 45.5 W to yield a mean SAR of 0.3 W/kg to match the level used by Toler et al. (1997) in their study of the same strain of tumor-prone mice chronically exposed to 435-MHz RFEMF. Decreases in mean SARs from increases in mouse weight with age during the exposure regimen were found to be much smaller than expected from considerations based on Durney et al. (1978), due to compensatory changes in the differential power absorptions between the mice and the cages.

Standard operating procedures were developed for all aspects of the study, from management and final necropsy of the mice, procedures compatible with those used by Toler et al. (1997). At the start, 250 weanling female mice were procured, and 13 were tested for quality control, which included necropsies and microscopic examinations. After a 10-day quarantine and review of the test results, the mice were found to be disease-free. Then, 200 mice were selected randomly and permanently marked for identification: 100 mice for RFEMF exposure and 100 for sham exposure. In addition, 25 mice were selected as sentinel animals for evaluations of health status, of which 10 were given complete necropsies and examinations.

RFEMF- and sham exposures (100 mice each) were started each day at 12:00 noon and terminated the next day at 8:00 am. During the 4-hour non-treatment periods on Mondays through Fridays of each week, 20 of the cages were changed each day and the 40 mice removed therefrom were weighed, palpated, visually inspected, and returned to clean cages.

Mice that had died spontaneously or had become moribund were completely necropsied and their tissues were fixed in 10% neutral buffered formalin and air-expressed to Pathology Associates International (PAI) for histopathologic evaluation. As part of standard operating procedure, PAI personnel returned to the facility at 6 months of treatment to perform health status evaluations and to supervise the scheduled necropsies of 5 sentinel mice. This procedure was repeated at 12 months of treatment. At the end of 18 months of treatment, the remaining mice were euthanized, and complete necropsies were performed by experienced PAI personnel without knowledge of the treatment received by each mouse. For a few mice, complete sets of tissues were not obtained because of autolysis or inherent difficulty in obtaining some tissues in section, such as the parathyroid gland.

Graphs of the mean weights of the RFEMF and sham groups versus weeks of treatment initially showed steep rises through about 15 weeks and more gradual rises thereafter, but the curves for the two groups were virtually coincident. Histopathologic examinations revealed the occurrence of neoplasms in various tissues, with mammary-gland carcinomas most frequent as expected. Single or multiplasms had occurred in 54 sham-exposed and 44 RFEMF-exposed mice; those had been diagnosed previously by palpation in 52 of the sham-exposed and all of the 44 RFEMF-exposed mice. By chi^2 test, the difference in tumor incidence between the two groups was not significant.

Plots of cumulative incidence of palpated and histologically-verified mammary tumors versus weeks of treatment of the RFEMF-exposed and sham-exposed groups were shown in Figure 2 of the paper. Up to about 55 weeks, the two plots were coincident. Beyond 55 weeks, the cumulative incidences in both groups rose more steeply, with a trend toward higher incidence in the sham group relative to the RFEMF group. However, a survival analysis of time-dependent differences in tumor onset showed no significant difference between the groups. The mean latencies to tumor onset were 64.0 ± 1.6 (SE) weeks for the RFEMF group and 62.3 ± 1.2 weeks for the sham group; by chi^2 test, the difference was not significant. A similar trend was found in mean tumor size versus time (Figure 3 of the paper): a slightly but nonsignificantly greater tumor-growth rate for the sham group.

The authors indicated that the tumors in several other organs or tissues were adequate in number to be analyzed. The numbers of mice in each group with neoplasms of the ovary, uterus, liver, and lung (by specific categories under each organ) were shown in Table 2 of the paper. The only significant difference between the groups was for lung tumors: alveolar-bronchiolar adenoma in 4 of 97 sham-exposed mice versus none in 99 RFEMF-exposed mice. Also discussed were the occurrences of various non-neoplastic lesions, which were ascribed mostly to aging in both groups.

The authors noted that the liver of several mice contained remnants of the intermediate stage of a tapeworm common to the domestic cat, the presence of which was postulated as due to fecal contamination of food or bedding from a cat at some stage of their manufacture. Exclusion of the affected mice from the analyses was shown to not alter the interpretation of the data on mammary neoplasm incidence. No evidence of other disease conditions was seen.

The authors used the product limit method of Kaplan and Meier (1958) to calculate the survival probabilities of the two groups. The results were plotted in Figure 4 of the paper as cumulative percent surviving mice versus time. Both groups had survival percentages of about 90% at 300 days and sharper declines to about 30% at 550 days, and with a slightly higher survival for the RFEMF group during the period 350-550 days.

The authors noted that the results of their study are consistent with those of Santini et al. (1988), Wu et al. (1994), and Toler and Shelton (1995) [meeting abstract] who had found that long-term exposure to low-level RFEMF did not promote cancer expression.

Malyapa et al. (1998a) performed a study subsequent to their investigations *in vitro* on RFEMF-induced DNA damage [Malyapa et al. (1997a, 1997b)]. As indicated in those earlier studies (discussed in the *in vitro* section below), they were unable to confirm the findings of Lai and Singh (1995) of single-strand-DNA breaks in the brains of rats exposed to 2.45-GHz RFEMF, and they endeavored to do so in this later study *in vivo*.

In this study, the authors exposed male Sprague-Dawley rats to 2.45-GHz CW RFEMF for 2 hours in a circularly-polarized cylindrical-waveguide system [Guy et al. (1979)] like that used by Lai and Singh (1995). The latter authors had used an incident power density of 2 mW/cm^2, for which the calculated nominal whole-body SAR was 1.2 W/kg. Malyapa et al. (1998a) measured temperature rises in the brains and hind quarters of rat cadavers induced by exposure to high RFEMF levels. They found that with the nose of the rat pointed toward the RFEMF source, the SAR in the brain was as much as twice that with the nose away from the source, so they also used 1.2 W/kg as the input power density, to duplicate the exposure conditions of Lai and Singh (1995) rather than attempting to experimentally verify the latter's dosimetry calculations.

Pairs of rats were concurrently treated for 2 hours, one of each pair RFEMF-exposed and the other sham-exposed. Either immediately or 4 hours after such exposure, the rats were euthanized, their brains excised and immersed in ice-cold Ames medium (composition similar to cerebrospinal fluid), and the cerebral cortex and hippocampus of each rat were processed for the alkaline comet assay and performed. A rat coding method was used to ensure that the entire procedure was done double-blind.

In the first series of experiments (4 rats per group), the rats were asphyxiated concurrently with CO_2 in a special chamber, and the brain of one rat of each pair was excised while the other rat remained dead in the chamber. In the second series (8 rats per group), one rat of each pair was asphyxiated and its brain excised before the other rat was euthanized. In the third series (8 rats per group), the rats were successively decapitated by guillotine and assayed similarly.

As a positive control, rats were irradiated *in vivo* with gamma rays from a ^{137}Cs source, at a whole-body dose of 0.5 Gy or 2 Gy at a rate of 0.82 Gy/min. They were then guillotined and cells from their cerebral cortex and hippocampus were processed blind for the alkaline comet assay, along with the cells from the RFEMF-exposed and sham-exposed groups.

The alkaline comet assay was performed essentially by the method of Olive et al. (1992), with some differences in media to prevent any DNA degradation in the lysis buffer during overnight lysis at 4°C. Electrophoresis was used at 0.6 V/cm for 25 minutes rather than 1 V/cm for 20 minutes. The comet images were viewed by fluorescence microscopy, digitized and stored, and each image was analyzed for "comet length" [CL] and "normalized comet moment" [NCM].

The pooled results for the rats asphyxiated concurrently, after which their brains were excised in sequence (first series), exhibited significant differences in CLs for the cortex or hippocampus that depended on the order of brain excision, specifically, the differences between the pairs of rats for which the brain of the sham-exposed rat was excised first versus the pairs for which the brain of that rat was excised second. The brain cells of the rats dissected first (either RFEMF- or sham-exposed) showed little DNA damage, whereas significant damage was observed for those dissected second. The authors noted that it took 3 to 4 minutes for death to occur by asphyxiation and at least 1.5 minutes to remove the brain and immerse it in the cold Ames medium, so they attributed the DNA damage to the elapsed time between death and brain removal. In addition, irrespective of RFEMF- or sham exposure, the DNA damage in the rats dissected second was greater than for the rats gamma-ray-exposed and guillotined.

In the second series, in which the time elapse between death and brain removal was about the same for all of those rats, the large difference in DNA damage found in the first experiment was absent. However, the pooled frequency distributions of the CL and NCM for the second series (shown as bar graphs of the counts per minute) indicated the presence of DNA damage in both RFEMF-exposed and

sham-exposed rats, but the SEs were comparable to their respective means and they overlapped (signifying non-significance).

Comparison of the results for the third series (rats sequentially guillotined) with those for the second series (rats sequentially asphyxiated) indicated that there were no significant differences in CL or NCM between RFEMF-exposed and sham-exposed rats regardless of euthanasia method or whether the cells were from the cortex or hippocampus. However, the frequency distributions of the CL and NCM differed for the two euthanasia methods.

The results for the gamma-ray irradiations and euthanasia by guillotine showed DNA damage (changes in both CL and CNM) for both 0.5 and 2 Gy. Also, the mean NCM for CO_2 asphyxiation by guillotine was comparable to the NCM for exposure at 0.5 Gy, but the CLs for the two euthanasia methods differed, primarily in their frequency distributions. Nevertheless, as noted by the authors, no differences in CL or NCM could be ascribed to the RFEMF exposure.

In the experiments in which the rats were euthanized 4 hours after RFEMF- or sham exposure, no significant differences were found in CLs, NCMs, or the frequency distributions thereof, either in the cortex or hippocampus, in contrast with the findings of Lai and Singh (1995). The possibility of tissue autolysis, raised for the latter study, does not appear to be relevant to the present study because in the latter, the rats were euthanized after the 4-hour time interval since the RFEMF- or sham exposures.

Investigation of the sensitivity to gamma radiation of the alkaline comet assay of Olive et al. (1992) used in the present study was described in a separately published paper by Malyapa et al (1998b). The authors noted that the technique was first described by Östling and Johanson (1984), and they discussed the differences between the version developed Singh et al. (1988) as modified further and used in the Singh et al. (1995) study, and the version developed by Olive et al. (1992). Also mentioned was that the NCM [normalized comet moment] and the CL [comet length] used to measure DNA damage were those described by Kent et al (1995).

The cell types used were rat lymphocytes in the G_0 (nondividing) phase and C3H 10T1/2 mouse fibroblasts in exponential growth [phases G_1 (65-70%), S (24-28%), and G_2 (8-10%)]. Test tubes containing appropriately treated lymphocytes were irradiated on ice with gamma rays from a ^{137}Cs source with doses of 0.6, 1, 3, and 5 cGy at a dose rate of 6.55 cGy/min, using one unirradiated test tube as a control. Monolayers of C3H 10T1/2 cells were irradiated either on ice or at 37°C with doses in the range 1-2 cGy at dose rates varied from 6.55 to 7.34 cGy/min, to keep the exposure times similar. Samples were processed for the comet assays, including a step for removal of any residual NaCl used in the cell-lysis step, and were coded to run the comet assays blind. All of the results were from three independent experiments, and were expressed as the average of the means from each experiment and the SEs. The statistical significance of the differences between irradiated and control groups was determined by Student's t-test.

Plotted for the lymphocytes on linear scales were the NCM in the range 0.4 to 2.0 and the CL in the range 20 to 45 μm, both versus dose in cGy, with SE bars. In each plot, the ordinate rose monotonically with dose, and the corresponding frequency distributions of NCM and CL broadened, with mean values increasing with dose. However, both plots displayed two linear regions of differing slope, with the slope in the dose range 0-1 cGy higher than for 1-5 cGy, indicating a biphasic dose-response relationship.

The CL values (μm) versus dose obtained by the method of Olive et al. (1992) and those by Singh et al. (1994) were plotted on the same graph, with the data for the latter normalized to 28 μm at 0 cGy. Using a visual best fit for the Singh et al. (1994) data, the data from both methods fitted the same line, but with increasing scatter versus dose, such as at the higher doses used by Singh et al. (1994). A linear dose-response was evident in a similar plot of normalized NCM.

The biphasic relationship at low doses, noted above in the present study, was evident in the experiments involving the irradiations of C3H 10T1/2 cells at 0°C and 37°C. Most of the CL and NCM data for treatment at 37°C were slightly smaller than treatment at 0°C, but the differences were not

significant, yielding the conclusion that for short exposure times (such as those used in this study), the irradiation temperature does not reduce the sensitivity of the comet assay significantly.

The authors remarked that a better detection sensitivity was achieved by their methods than by the method used by Singh et al. (1994).

4.2 CANCER INDUCTION AND PROMOTION IN MAMMALIAN TISSUES (*IN VITRO*)

A number of studies have been directed specifically toward possible malignant transformations of cells by exposure to RFEMF *in vitro*. In addition but perhaps of peripheral relevance to RFEMF as a possible *in vivo* carcinogen are studies of its presumed effects as a mutagen. Both kinds of studies are analyzed below in approximate chronological order.

Heller (1970) investigated whether exposure to pulsed RFEMF could cause chromosomal damage to cells in mitosis by stimulation with a mitogen. Human lymphocytes separated from peripheral circulation were cultured for two days with the mitogen phytohemagglutinin (PHA) and then exposed for 30 minutes between electrodes spaced 2 cm apart to 21-MHz pulsed RFEMF (10-μs pulses, 100 pps) at 500 V/cm (50 kV/m) peak-to-peak. The free-space equivalent average power density was 83 mW/cm^2. The frequency and other RFEMF characteristics were selected as most effective, based on several citations of damage in garlic-root cells from exposure to frequencies in the range 5-40 MHz. The culture temperature was maintained at 27°C during exposure.

Cell cultures were fixed post-exposure either immediately or after recovery periods of 24 to 36 hours before fixation. Standard air-dry films were stained and scored for the following types of chromosomal abnormalities: chromosomal breaks, dicentric chromosomes, chromatid breaks, endoreduplication figures, acentric fragments, and polyploid number chromosomes. The results were presented in Table 1 of the paper. In the control culture, 600 cells were scored: found were chromosome breaks in 6 cells, acentric fragments in 6 cells, and polyploid chromosomes in 4 cells. The overall result was a mean of 0.016 abnormalities per cell. In the culture fixed right after exposure, 500 cells were scored, of which 4 cells had chromosome breaks, 2 cells chromatid breaks, 6 acentric fragments, and 12 cells with polyploid chromosomes. The overall mean was 0.036 abnormalities per cell, with a chi^2 value of 5.2 and $P=0.02$ relative to the control culture. In the culture fixed 24 hours post-exposure, 2000 cells were scored, in which all six types of chromosomal abnormalities were found, with a mean of 0.056 abnormalities per cell, a chi^2 value of 16 and $P<0.01$ relative to the control culture. A total of 850 cells in the culture fixed 36 hours post-exposure were scored, which yielded a mean of 0.077 abnormalities per cell, a chi^2 value of 24 and $P<0.01$ relative to the control culture.

It appears that only one cell culture was treated for each experimental condition. Not stated was the reason for selecting different numbers of cells for scoring. Also unclear is when was the control culture fixed following the two-day culturing with PHA. If this was done immediately, then comparing the results for the two post-exposure delay periods with those for the control culture are open to question, i.e., no comparisons were made between RFEMF-exposed and control cultures fixed at corresponding times. In addition, it appears that the control culture was not sham-exposed.

The author also performed similar experiments with cultured lung cells from the Chinese hamster, and stated that the RFEMF had produced significant numbers of chromosomal abnormalities, but presented no results other than a few presumably representative microphotos.

Stodolnik-Baranska (1974) investigated what effects exposure to pulsed 2.95-GHz RFEMF would have on human peripheral-lymphocyte suspensions without and with stimulation by the mitogen PHA. In the first of two series, cultures without PHA were exposed to pulsed RFEMF (1-μs pulses at 1200 pps) with a horn antenna at 7 mW/cm^2 average power density for 4 hours daily or at 20 mW/cm^2 for 15 minutes, each for 3 or 5 days. In the second series, after 66 hours of incubation with PHA, lymphocyte cultures were exposed at 20 mW/cm^2 for 5, 10, 15, 20 minutes or for two 20-minute durations 30 minutes apart. Other cultures were exposed at 7 mW/cm^2 for 3 or 4 hours either once or for 4 hours daily for 3 days, and still others were exposed for 10 minutes after incubation for 0, 59, 70, or 71.5 hours. In each

experiment, the cultures were fixed, dried, and stained. The temperature of the culture medium stayed constant at 37°C during 4 hours of exposure at 7 mW/cm^2; at 20 mW/cm^2, it increased by 0.5°C after 15 minutes and by 1°C after 20 minutes. The numerical results of the experiments were tabulated.

At 20 mW/cm^2, the mitotic index (MI) rose monotonically with exposure duration from 12.0% at 0 minutes (no exposure) to 25.0% for 40 minutes (the two 20-minute periods). The rises for exposure durations of 20 and 40 minutes were significant at the 95% confidence level relative to the expected MI ranges. The variations of the MIs for 10 minutes of exposure at 20 mW/cm^2 versus the number of hours of incubation were non-monotonically higher than the variations for the unincubated control, but each was within its corresponding expectation range.

Tabulated at 20 mW/cm^2 versus exposure duration for 5, 10, 15, and 20 minutes were the numbers of chromosomal aberrations termed "dicentrics", "hyperploidy", "hypoploidy", and "breaks". Non-monotonic increases were seen in all four types, with the largest increase for chromosomal breaks. The specific values were 2.0 (unexposed control), 1.0 (5 minutes), 7.0 (10 minutes), 5.0 (15 minutes), and 28.0 (20 minutes). Statistical analysis of these results was not presented. The observation that the increases were not monotonic may indicate that uncontrolled non-RFEMF factors may have been present, e.g., related to culture-temperature variations at 7 mW/cm^2 and the observed temperature increase with exposure duration at 20 mW/cm^2.

Chen et al. (1974) grew cultures of Chinese-hamster cells in liquid medium within plastic containers, each of which was placed in a matched, open-ended waveguide for exposure to 2.45-GHz CW RFEMF. The temperature in the waveguide was controlled with an electric heater, and the culture temperature was monitored continuously by thermocouple during exposure. After exposure at specified RFEMF levels and durations, the cells were permitted to grow for 24 hours, after which they were replanted in fresh medium and again allowed to grow for 24 hours. At this time, their growth was stopped at the metaphase stage and the cells were swelled, fixed, placed on slides, and stained for chromosome analysis, consisting of determining the percentages of eleven types of chromosomal aberrations and the percentage of mitotic cells. Control cultures were sham-exposed but otherwise treated similarly.

In one set of experiments, the cultures were initially at 22°C (room temperature) and their temperatures at exposure end were noted. (Presumably, the waveguide temperature was not controlled with the heater.) Cultures were exposed at 50 mW/cm^2 for 10 minutes, at the end of which their temperature was 37°C. Other cultures were exposed at 85 mW/cm^2 for 4, 8, or 10 minutes, which produced final temperatures of 37°C, 40°C, and 41°C.

As noted by the authors, there was no significant difference in the mean number of cells with all types of aberrations between exposed and control cultures, but for some exposure conditions, the mean number of cells with specific aberrations was significantly higher than for the control cultures. As examples, 8% of the cells in the culture exposed at 85 mW/cm^2 for 8 minutes showed chromatid breaks versus only 1.8% of the control cells; and polyploidy, absent in the controls, was found in 2% of the cells exposed at 85 mW/cm^2 for 10 minutes. However, the percentages of chromatid breaks in cultures exposed at 50 or 85 mW/cm^2 for 10 minutes were both smaller than for controls. Also, polyploidy was found in 0.2% of the cells exposed at 85 mW/cm^2 for 4 minutes, but was absent in the cells exposed at 85 mW/cm^2 for 8 minutes. Thus, no clear dose-dependence was evident in these results.

In a similar set of experiments, cultures were initially heated to 37°C in an incubator and the waveguide temperature was held at 37°C during exposure. The exposures were at 20 mW/cm^2 for 8 or 10 minutes, 50 mW/cm^2 for 4 minutes, or 85 mW/cm^2 for 2 or 3 minutes. The respective final culture temperatures were 40°C, 41°C, 42.5°C, 40.5°C, and 43°C. The results were qualitatively similar to those for the previous set, with again no clear dose-dependence. One interesting observation regarding the two sets was that 93% of the control cells in the first set were in mitosis versus only about 15% in the second set and that in neither set did the various exposure conditions markedly alter these percentages. Not presented was the methodology used in measuring and controlling culture temperature during RFEMF exposure.

Chen et al. (1974) also did experiments similar to the first set, but with human amnion cells. In general, the results showed smaller percentages of chromosomal aberrations than for the Chinese-hamster cells, but otherwise were again qualitatively similar.

Assessing the findings of this paper was difficult because of inadequate statistical treatment of the results. Specifically, the authors presented the mean percentage and standard deviation (SD) for each chromosomal aberration in the sham-exposed controls, but gave the mean percentages for the RFEMF-exposed cultures without SDs, which were not calculated "in the interest of saving time". Moreover, even for the controls, the SDs were comparable to, and in some cases exceeded, the respective means. Because of the large variabilities in the results and the absence of dose-dependence, little if any credence can be given to the authors' suggestion that exposure to RFEMF induces chromosomal abnormalities to a significant degree.

Brown and Marshall (1986) sought nonthermal effects of RFEMF exposure on the growth and differentiation of the murine erythroleukemic (MEL) cell line. They noted that in response to an inducer such as hexamethylene bisacetamide (HMBA), MEL cells form hemoglobin and exhibit other kinds of erythroid differentiation. Before RFEMF exposure, the proliferation and differentiation response to HMBA of MEL-cell cultures were characterized in 60-mm or 100-mm dishes at 37°C in a humidified atmosphere of 5% CO_2. For exposures, 12-ml cultures initially having 600,000 MEL cells in 3 mM of HMBA were cultured in thin-wall, stoppered, RFEMF-transparent cellulose nitrate tubes as vessels. Cell proliferation was monitored with a hemocytometer. Hemoglobin-bearing differentiated cells were detected by staining with a benzidine reagent. The cells were lysed to release hemoglobin, and the supernatant hemoglobin was assayed and expressed relative to the total supernatant, using bovine albumin as the standard for comparison.

For each experiment, two tubes of HMBA-cultured MEL cells were exposed for 48 hours to 1.18-GHz CW RFEMF at power densities of 5.5, 11, and 22 mW/cm^2 in an anechoic chamber that had a duct through which temperature-controlled air was forced. Two control tubes were incubated in a 37.4°C water bath. The temperature of the culture medium was monitored continuously with a Vitek probe inserted into the stopper of one each of the exposed and control tubes. The SARs were determined by measuring the cooling rates with the Vitek probe immediately after RFEMF exposure and were verified by calorimetry. Normalized SAR was 3.32 W/kg per mW/cm^2 for two concurrently exposed tubes. The SARs at the stated input power densities were 18.5, 36.3 W/kg, and 69.2 W/kg with the incubation temperature held at 37.4°C. Control cultures were held at the same temperature in a water bath.

Four replicate experiments were performed at each of the three RFEMF levels. The growths of RFEMR-exposed and control cultures were compared by measuring the elapsed times for the cells to double in number. Cell differentiations were compared by counting the percentages of cells stained by a hemoglobin-specific dye and by determining the amounts of hemoglobin produced. The mean results at each RFEMF level for each endpoint (cell-doubling time, differentiation as determined by the percentage of benzidine-positive cells and by the hemoglobin content) did not differ significantly from their respective controls. Also, the mean values of each endpoint at the three RFEMF levels did not differ significantly from one another.

The finding of no RFEMF effect in this study is highly credible in view of the well-detailed experimental protocol and the accuracy and precision of the RFEMF dosimetry.

Balcer-Kubiczek and Harrison (1985) studied the presumed carcinogenic activity of 2.45-GHz RFEMF in combination with benzo[a]pyrene (BP) or X-rays in C3H/10T1/2 mouse-embryo fibroblasts. They also assessed the effects of the tumor promoter 12-O-tetradecanoylphorbol-13-acetate (TPA) (in a non-cytotoxic and non-transforming concentration) on the transformation induction of cells exposed to either RFEMF, X-rays, or both.

Cultures of C3H/10T1/2 cells maintained in Eagle's basal medium with 10% fetal bovine serum and the antibiotic gentamycin were grown at 37°C and seeded into 25-cm^2 plastic flasks as monolayers at a surface density 10,000 cells/cm^2 per flask. Far-field exposures to 2.45-GHz RFEMF were done in an

anechoic chamber that embodied a horn antenna directed toward a water bath held at 36°C, into which each monolayer-bearing flask was immersed to a depth of about 2.5 cm. A 25x25-cm quarter-wave impedance-matching plate was placed between the horn and the surface of the bath. BP-treated cells in dimethylsulfoxide (DMSO) at a final concentration-dilution in the medium to 0.12% were exposed to the RFEMF for 24 hours at an SAR of 4.4 ± 0.8 W/kg, determined by calorimetry and local-field measurements, Such exposure increased the steady-state medium temperature by a maximum of 1.2°C. Other cells were exposed to the RFEMF for 6 hours, followed by irradiation with X-rays for a total dose of 1.5 or 4.5 Gy at a dose rate of 0.39 Gy/min, RFEMF-exposed again for 18 hours, and treated with TPA right after irradiation. [Gy stands for "gray", a unit of radiation absorption dose (rad) equal to 100 ergs per gram of tissue.] As controls, cells were sham-RFEMF-exposed and treated with the same doses of X-rays or BP, with or without subsequent incubation with TPA in acetone (at a final concentration dilution in medium of 0.05%).

On treatment completion, the cells were washed, trypsinized and counted, appropriately diluted, and assayed for survival or transformation by plating 100 or 300 viable cells on large (100-mm) dishes. The cells for survival assay (by colony formation) were incubated for 2 weeks, and those for transformation assay (scored for aberrant clones) were incubated for 6-8 weeks. About 130 dishes were used for assaying transformation frequency for each treatment, calculated from the mean numbers of transformants per plate and the fraction of dishes without transformed colonies. The transformation frequency per viable cell was determined from the average number of transformants per plate and the average number of surviving cells per plate.

Shown in Table 1 of the paper were the pooled results for mean plating-efficiency percentages and SEs in the absence of the RFEMF, without TPA (12 groups) and with TPA (4 groups). The respective percentages were 22.8 ± 2.1 and 29.0 ± 0.6. Also shown were the percentages for RFEMF alone (16 groups) and for RFEMF + TP (4 groups), respectively 9.7 ± 0.6 and 13.7 ± 0.9. Stated in a footnote to the table was that the pooling included groups with solvent (0.05% acetone post-treatment) or 0.12% DMSO during RMEMF exposure.

Plots of normalized mean cell-survival fractions and SEs (on an exponential scale) versus BP concentrations (on a linear scale) for flasks treated only with BP or RFEMF-exposed and concurrently treated with BP were displayed in Figure 2 of the paper; the normalization was relative to the DMSO/no-RFEMF control. As semi-exponential plots, the curves were approximately parallel in the decreases with increasing BP concentration, with the survival fractions for BP alone about twice those for treatment with both BP and RFEMF. The points for mean survival-fraction at zero BP concentration on the two curves were about 1 and 0.5. These results possibly implied that the roughly 2:1 spacing between the curves was due to the RFEMF, and that the increase in BP concentration contributed to the survival-fraction decrease in each curve in a similar manner.

Semi-exponential plots of normalized mean cell-survival fractions versus X-ray dose were presented in Table 3 of the paper (without SEs). The upper curve was for cells treated with X-rays only, with and without TPA, and the lower one was for cells treated with X-rays + RFEM, also with and without TPA. Again, the two curves were parallel to one another. In each curve, there were only small differences (probably non-significant) between the use of TPA and its absence. However, the curve for X-rays + RFEMF was lower than the curve for X-rays only by more than a ratio of 2, apparently indicating that the RFEMF was the primary factor in the difference between the curves.

Figure 4 of the paper was a semi-exponential plot of the normalized mean transformation frequency (with SEs) for BP alone and for BP + RFEMF, versus BP dose in the range 1.25-12.5 μM. The normalization was relative to the plating efficiency in the matching control groups. Both sets of points fitted on the same curve, leading the authors to conclude that the RFEMF had no effect on the transformation frequency in the subpopulation of the cells that survived treatment. At one BP dose (5 μM), however, the point for BP alone was higher than the point for BP + RFEMF, and their SE bars did not overlap, possibly indicating that the mean transformation frequency for BP alone at that dose was significantly higher than for BP + RFEMF. Figure 5 showed a similar plot of mean transformation frequency versus X-ray dose for X-rays alone and for X-rays + RFEMF, also normalized to plating

efficiency in the matching control groups. There was no significant difference between the two points at each X-ray dose.

The authors remarked in their discussion that their data showed a significant enhancement of transformation frequency in C3H/10T1/2 cells exposed to RFEMF [at 4.4 W/kg] and X-rays and subsequently cultured with the promoter TPA. They also noted that exposures to RFEMF in combination with BP or X-rays without TPA had no such effect. They concluded that the RFEMF modified the cell transformation initiated by the X-rays and promoted by the TPA.

The display of ratios in the curves, which were presented without any experimental data from the use of the specific agents investigated or experimental data for the control cultures used to normalize the experimental data, renders the validity of the findings of this study obscure. In particular, not presented were the numerical values of the means and SEs for the controls in each experiment. If the control SEs were large, they would indicate the probable presence of uncontrolled confounders.

In a subsequent short communication, Balcer-Kubiczek and Harrison (1989) noted that because the positive results of their previous study were observed "in a rather involved experimental protocol, our findings are difficult to interpret in terms of biohazards from microwave exposure". This later study was directed toward testing whether prolonged exposure to RFEMF could elicit and/or enhance effects of other known carcinogens. The exposure system was described in Harrison et al. (1985). (It was also used in the previous study but not cited therein.)

The main protocol and the results thereof were displayed in Table I of the paper in more detail than in the previous study. However, the presentation of the data was obscure. The authors remarked that there were no significant differences in the weighted mean plating efficiency among cells RFEMF-exposed [at 4.4 W/kg] and those sham-exposed and either subsequently cultured with TPA at 0.1 μg/ml or not treated with TPA. Also, no significant differences were seen in mean survival rates for cells exposed to X-rays alone [at 1.5 Gy] or cultured with TPA after X-ray exposure. As noted by them, these results differed from their previous findings.

Presented in Figure 1 of the paper were bar graphs (and SEs) of the mean transformation frequency for cells that were exposed as follows, with all cells TPA-treated post-exposure: RFEMF-exposed only, X-ray-exposed only, exposed to X-rays before RFEMF exposure, and exposed to X-rays after RFEMF exposure. Also displayed were: bars for similarly exposed cell groups not treated with TPA, controls not exposed to either the RFEMF or the X-rays, controls treated only with TPA in its solvent (acetone), and (for each exposure group) the groups treated with the solvent only rather than with TPA.

The mean transformation frequency for the TPA-RFEMF-only group was somewhat higher than for the TPA-only group, but was about the same as those for the three exposure groups treated with acetone only. The mean transformation frequencies for the three TPA groups (X-rays only, X-rays + RFEMF, and RFEMF + X-rays) were much higher than for the TPA-RFEMF-only group, with the X-ray + RFEMF and RFEMF + X-ray groups higher than for the TPA-RFEMF-only group. However, the error bars for all three of those groups overlapped, possibly indicating non-significant differences among them.

From the results above with TPA, the authors concluded that the RFEMF alone initiated cell transformation (relative to the sham-exposed controls), the X-rays alone promoted much higher transformation than the RFEMF, and the two combinations of RFEMF and X-rays promoted somewhat higher transformation frequencies than the X-rays alone. The latter point is questionable because of the overlapping error bars mentioned above, i.e., the RFEMF added little if any to the effects of the X-ray exposures. Not clear was why the acetone-only results were comparable to one another and to those of the RFEMF-TPA group.

In a third study, Balcer-Kubiczek and Harrison (1991) investigated whether RFEMF exposure of mouse-embryo-fibroblast-cell [C3H/10T-1/2] cultures would induce malignant transformation in such cells. They exposed cultures for 24 hours to 2.45-GHz RFEMF (amplitude-modulated at 120 Hz) alone at an SAR of 0.1, 1, or 4.4 W/kg, or to the RFEMF at 4.4 W/kg before or after exposure to X-rays at 0.5, 1, or

1.5 Gy. Control cultures were sham-exposed. After such treatments, cultures with or without incubation with the tumor promoter TPA at 0.1μg/ml were assayed for the incidence of neoplastic transformations by counting the number of transformed foci in culture dishes.

The sham-exposed cultures exhibited low incidences of neoplastic transformation, and the cultures incubated with TPA showed a slightly higher mean incidence than those not incubated with TPA. A plot of mean neoplastic transformation incidence (linear scale) versus SAR (exponential scale) for the RFEMF-exposed cultures not incubated with TPA showed basically no differences from sham-exposed cultures or any changes with increasing SAR, indicating that the RFEMF alone did not promote transformation. However, the mean neoplastic transformation incidence rose with SAR for the RFEMF-exposed cultures that were incubated with TPA. The authors interpreted those results as indicating that RFEMF acts synergistically in a dose-dependent manner with TPA to promote neoplastic transformation.

In graphs of mean neoplastic transformation incidence versus X-ray dose (Figure 2 of the paper), the cultures not incubated with TPA displayed a relatively small rise with X-ray dose (0, 0.5, 1.0, 1.5 Gy), independently of whether the cultures were exposed to RFEMF (at 4.4 W/kg) or sham-exposed. For the cultures incubated with TPA, however, the mean transformation incidence rose linearly with X-ray dose for those exposed to 4.4 W/kg, and also linearly for sham-exposed cultures, but the latter cultures had about half as many mean incidences as the 4.4-W/kg cultures at corresponding X-ray doses. Thus, the exposure at 4.4 W/kg of cultures treated with X-rays plus TPA appeared to increase neoplastic transformation incidence relative to sham exposure plus TPA, but the differences in incidence at corresponding X-ray doses were statistically significant only for 0 and 0.5 Gy.

For cultures that were not treated with X-rays, sham exposure yielded low incidences of neoplastic transformation, with a slightly higher mean incidence for those incubated with TPA than those not incubated with TPA. Thus, TPA alone (at the dose used) did not promote transformation. On the other hand, RFEMF-exposed cultures not incubated with TPA showed essentially no differences from sham-exposed cultures or any significant changes with increasing SAR. Thus, the RFEMF alone also did not promote transformation. However, for the RFEMF-exposed cultures incubated with TPA, the mean neoplastic transformation incidence increased with SAR, an apparent indication that the RFEMF acted synergistically in a dose-dependent manner with TPA to promote neoplastic transformation. The numerical results were as follows:

a. Found were 14 foci in 1494 dishes of sham-exposed cultures incubated with TPA, and 4 foci in 887 dishes of sham-exposed cultures not incubated with TPA.

b. Also found were 48 foci in 704 dishes of cultures exposed at 4.4 W/kg incubated with TPA, but only 4 foci were found in 800 dishes exposed at 4.4 W/kg not incubated with TPA.

Several aspects of this investigation make interpretation of the findings difficult and uncertain. First, a plot of mean neoplastic transformation incidence versus SAR (Figure 1 of the paper) indicated an apparently linear rise of incidence with SAR (points at 0.1, 1.0, 4.4 W/kg). That result can be misleading because, unlike what was done in plotting incidence versus x-ray dose (Figure 2 of the paper), in which linear scales were used for both variables, the authors used a linear scale for incidence and an exponential scale for SAR. If the three SAR points had been plotted on a linear scale also, the graph would have displayed a fivefold sharper rise with SAR between 0.1 and 1.0 W/kg than between 1.0 and 4.4 W/kg.

The numbers of foci found relative to the numbers of dishes treated were small. The numbers of dishes used differed considerably for each treatment (see paragraphs a. and b. above). This point raises the question whether the authors may have increased the number of dishes for each treatment until adequate percentages of foci for statistical analysis were obtained. Also open to question is how well was culture temperature controlled, particularly at 4.4 W/kg.

Based on the foregoing analyses of the three studies by Balcer-Kubiczek and Harrison [1985, 1989, and 1991], little if any credence can be given to their findings.

Garaj-Vrhovac et al. (1992) remarked: "Microwave radiation is one of the known mutagens which cause changes at the level of DNA synthesis and its structure." Based on this premise, they exposed samples of human whole blood to 7.7-GHz CW RFEMF and analyzed lymphocyte samples therefrom for chromosomal aberrations. They also counted the micronuclei in the lymphocyte samples, to determine the origin and relationship of micronuclei to specific aberrations produced by RFEMF exposure. The blood donors were stated to be non-smoking healthy persons, with no recent diagnostic or occupational exposure to either ionizing or non-ionizing radiation.

The exposure arrangement was described in Garaj-Vrhovac et al. (1990b). Each sample was placed horizontally on a thin Bakelite sheet supported on 1.2-meter-high blocks. An applicator consisting of "an optimal horn antenna" with an 80-cm slit was placed just above the sample for downward irradiation. The RFEMF source was a reflex klystron that fed the horn through an isolator, a variable attenuator, and instrumentation for measuring the RFEMF frequency, forward power, and the standing-wave ratio (SWR). The authors stated that the SWR did not exceed 1.08 with or without cell samples present, but the sample holder was not described. They also noted that the electromagnetic field distribution at the slit cross section was calculated theoretically and checked experimentally, and that sample temperature was measured with a digital contact thermometer, but they provided no data.

The blood samples were exposed to 7.7-GHz RFEMF at 0.5 or 10 mW/cm^2 for 30 minutes, or at 30 mW/cm^2 for 10, 30, or 60 minutes, all at 22°C constant temperature. Those power densities were selected to span 10 mW/cm^2, which the authors characterized as the ANSI exposure limit (no date), apparently unaware that the 10-mW/cm^2 limit in the 1974 ANSI guidelines had been superseded twice since then (in 1982 and 1991).

After exposure, lymphocytes were cultured for 48 hours with the mitogen phytohemagglutinin (PHA) and incubated with colchicine during the last three hours. The cell samples were then fixed and examined for chromosomal damages that were characterized as acentric fragments, dicentric and ring chromosomes, or chromatid and chromosome breaks. In each experiment, 500 metaphases were analyzed. In preparations from fixed and stained samples of 1000 bi-nuclear cells with well-preserved cytoplasm, the numbers of cells that had 0, 1, 2, or 3 micronuclei [fragments of the cell genome] were determined for each exposure level. The total number of cells affected at that level was tabulated as a percentage of the total number of cells examined for that level. Tabulated also were the percentages of cells with each type of chromosomal aberration and the total percentages thereof for each exposure level. Similar data for control cells were presented for comparison. Not indicated was whether the control samples had been sham-exposed.

For the controls, 991 of the 1000 cells examined had no micronuclei, and the other 9 had only 1 micronucleus (0.9%). Exposure at 0.5 mW/cm^2 for 30 minutes yielded 986 cells with no micronuclei and the other 14 cells with only 1 micronucleus (1.4%), a nonsignificant difference ($p>0.05$) relative to the controls. The results at 10 mW/cm^2 for 30 minutes and at 30 mW/cm^2 for 10 minutes were comparable to one another, respectively 3.6% and 4.0%, and both were nonsignificantly different from controls. However, at 30 mW/cm^2 for 30 and 60 minutes, the results were 4.4% and 6.8% respectively; the differences from controls were both significant ($p<0.05$ and $p<0.001$).

Regarding chromosomal aberrations, 0.5% of the control cells exhibited chromatid breaks, another 0.5% had chromosome breaks, still another 0.5% had acentric chromosomes, and the remainder no aberrations, for a total of 1.5%. The corresponding results for exposure at 0.5 mW/cm^2 for 30 minutes were: 1.1%, 0.5%, and 1.1%, for a total of 2.7%, a nonsignificant difference. At 10 mW/cm^2 for 30 minutes and at 30 mW/cm^2 for 10 minutes, the aberration counts were 5.5% and 4.9%, both significantly higher ($p<0.0001$) than for controls; the results at 30 mW/cm^2 for 30 and 60 minutes were still higher: 6.1% and 7.2% respectively ($p<0.001$).

The authors displayed those results as bar graphs (without variances) and noted that the number of acentric and dicentric chromosomes for each exposure regimen and the number of micronuclei were correlated. In their discussion, however, they also remarked that there was no positive correlation

between total chromosomal aberrations (including chromatid and chromosomal damage) and the occurrence of micronuclei.

Their overall conclusion was: "The frequency of structural chromosomal damages and microneuclei increases depending on the applied power density and duration of exposure (1.4% to 6.8%) which is another proof of the mutagenic effect of microwave radiation."

More detailed information about many aspects of this study would be needed to assess the degree of validity of its findings. Important points not discussed were whether the control samples were sham-exposed and whether the results for RFEMF-exposed samples from each subject were compared with control samples from the same subject. Also absent were experimental data on: how well the temperature within the cell preparations were controlled during RFEMF exposure, the size of the spatial temperature variations within samples, and measurements of incident power densities and their spatial variations. Thus, the presence of artifact cannot be ruled out. In addition, the numbers of cells affected relative to the totals examined were small, details about statistical treatment of the data were not given, and the difference in the total chromosomal aberrations for exposure at 10 mW/cm^2 and 30 mW/cm^2 was slight. These results raise the question of whether the reported effects were dose-dependent. For these reasons, little if any credence can be given to the findings of this study.

Ivaschuk et al. (1997), in one of three papers from the same organization, investigated whether rat PC12 cells treated with nerve growth factor (NGF) and exposed to time-domain-multiple-access (TDMA)-modulated 836.55 MHz-RFEMF would affect expression of the genes that encode *c-jun* and *c-fos*. That cell line was selected because it is highly responsive to NGF. Monolayer cultures of cells in an appropriate medium were seeded onto 100-mm Petri dishes to a depth of 3 mm. The cultures were treated with 50 ng/ml of NGF just before RFEMF exposure.

The exposure system consisted of a specially configured TDMA transmitter at carrier frequency 836.55 MHz that fed, through an in-line wattmeter, to a power splitter with four outputs. The TDMA transmitter frequency and modulation conformed to the North American digital cellular telephone standard. When the carrier is on, it is not amplitude-modulated (pulsed); rather it consists of digital data of constant amplitude, with the phase of each bit shifted by ± 45° or ± 135° relative to the preceding bit as determined by the data.

Two of the output ports of the splitter were connected via RG-58 coaxial cables of equal length to transverse-electromagnetic (TEM) cells of square cross section, with a 50-ohm load terminating each cell. One of the TEM cells was used for RFEMF exposures and the other for concurrent sham exposures. A third splitter port served for monitoring the RFEMF power levels. It was connected through a bidirectional coupler and an attenuator to a diode detector. The forward-power port of the coupler was connected through a thermistor mount to a power meter, the analog output of which was connected to a chart recorder. (The fourth splitter port was not used.)

The slot power densities used were 0.09, 0.9, and 9 mW/cm^2 with the carrier on, and the carrier was active for 6.67 milliseconds per 20-millisecond frame, for a duty cycle of 33%., thus yielding time-averaged levels of 0.03, 0.3, and 3 mW/cm^2 . In most of the experiments, two stacks of two Petri dishes each were placed centrally on the septum of each TEM cell (with the E-field normal to the dishes); in a few others, dishes were placed both on the septum and the bottom of each cell. The dosimetry aspects were based on Burkhardt et al. (1996). With 0.9 mW/cm^2 (0.51 W input power), calculations for the septum-only conditions yielded a spatial average SAR of 2.6 ± 1.9 [SD] μW/g or 2.9 μW/g per mW/cm^2. The single-dish minimum SAR for dishes farthest from the septum was 0.5 μW/g and the single-dish maximum SAR for dishes closest to the septum was 4.6 μW/g. Unclear was whether those SARs were calculated for the 33% or 100% duty cycle. Presuming the latter, the time-averaged SARs were one-third of the indicated values, e.g. a time-averaged spatial mean of 097μW/g. Exposures at slot levels of 0.09, 0.9, and 9 mW/cm^2 were for total durations of 20, 40, or 60 minutes with 20-minute on/off intervals for the latter two durations, to simulate intermittent use of cellular telephones.

Cell-media temperatures were measured with a thermometer using a microprocessor-controlled Vitek-type-probe that could resolve temperature changes as small as 0.002°C. The authors indicated that no temperature rise was detectable at any power density used. Also determined were the local static magnetic field (31 ± 12 μT) and the ambient 60-Hz magnetic field [0.13 ± 0.02 μT (rms) at the TEM cell used for RFEMF exposure and 0.20 ± 0.04 μT (rms) at the TEM cell used for sham exposure].

After treatment, the Petri dishes were removed in random sequence from the TEMs for analysis, thereby yielding data from the dishes at all locations. On dish removal, the RNA of each was isolated, size-fractionated on formaldehyde/agarose gels, and transferred to MSI Magna nylon membranes by positive pressure as "Northern blots", cross-linked with ultraviolet at 0.6 nW/cm^2 for 5 minutes in a buffer containing cDNA probes for *c-fos* and *c-jun*, and labeled with 32P at 3,000 Ci/mole. GPDH (glyceraldehyde-3-phosphate dehydrogenase) was used as an internal standard because it was found to be unaffected by RFEMF exposure.

Following three washings, the blots were evaluated with an Ambis 4000 radioanalytic imager and accompanying software. The cDNA probes recognized mRNAs of 2.2 kb for *c-fos*, 2.7 and 3.2 kb for *c-jun*, and 1.4 kb for GPDH. In each experiment, variations in the GPDH band intensity were generally ± 15-20%. For each RFEMF-exposed or sham-exposed sample, the results were expressed as ratios of the dpm (radioactive disintegrations per minute) for *c-fos* or *c-jun* to the dpm for GDPH, and then as the ratios of those ratios (E/C). The analyses were done without knowledge of sample identity.

The authors indicated that *c-fos* expression peaked at 15-30 minutes after NGF stimulation and then returned to baseline by 60 minutes. They also noted that it was possible to measure the levels of *c-fos* mRNA only after the first 20 minutes of exposure, because the levels had returned to baseline after a second 20-minute exposure. Thus, the E/C results for *c-fos* expression in five experiments after RFEMF exposure for 20 minutes at 0 (sham/sham ratio), 0.09, 0.9, or 9 mW/cm^2 were displayed in Table 1 of the paper, together with their respective means (and SDs). The sham/sham E/C values for the five experiments ranged from 0.80 to 1.15, with a mean of 0.99 ± 0.14. The mean values for 0.09 and 0.9 mW/cm^2 were essentially the same: 1.03 ± 0.15 and 1.03 ± 0.11, respectively. For 9 mW/cm^2, the E/Cs ranged from 0.58 to 1.01, with a mean of 0.86 ± 0.17. None of those means differed significantly from 1.00.

Concerning *c-jun*, the authors noted that the dynamics of its expression differed from that for *c-fos* in peaking 30-45 minutes after NGF stimulation and returning to baseline by 120 minutes. Accordingly, they reported the *c-jun* results from exposure for 20, 40, or 60 minutes at the same four levels of RFEMF, which were displayed in Table 2 of the paper. For the five experiments at 0, 0.09, and 0.9 mW/cm^2 and the three durations, the mean E/Cs also did not differ significantly from 1.00. This was also true with 9 mW/cm^2 for durations of 40 and 60 minutes, but the mean E/C for 9 mW/cm^2 and 20 minutes was 0.61 ± 0.11, significantly less than unity ($P<0.05$ ANOVA).

The authors concluded that exposure of NGF-treated PC12 cells to TDMA-modulated RFEMF at mean intensities of 0.09, 0.9, or 9 mW/cm^2 does not alter the expression of the early response genes *c-fos* or of *c-jun* at 0.09 or 0.9 mW/cm^2, but that a 39% decrease in *c-jun* transcript level was observed after exposure to 9 mW/cm^2 for 20 minutes. The authors also noted that the lack of effect of RFEMF exposure on *c-fos* expression was found by Phillips (unpublished data) in NGF-treated PC12 cells exposed to 60-Hz magnetic fields up to 100 μT, in contrast with Phillips et al. (1992). The latter authors reported observing large *c-fos* increases in CCRF-CEM T-lymphoblastoid cells exposed at 100 μT.

Stagg et al. (1997), in the second paper, investigated whether exposure of rat glioma cells to TDMA-modulated 836.55-MHz RFEMF would promote tumor growth by altering DNA synthesis and elevating cell proliferation. Normal glial cells were also studied. The RFEMF exposure system consisted of two TEM cells [(like those used by Ivaschuk et al. (1997)] within standard incubators that had a 5% CO_2 atmosphere and were held at 37°C. One TEM cell was used for sham exposure and the other for RFEMF exposure. Exposures were done at slot power densities of 0.09, 0.9, or 9 mW/cm^2, with 33% duty cycle.

C_6 glioma cells were obtained from an outside supplier and grown in F-10 medium supplemented with 15% horse serum and 2.5% fetal bovine serum (FBS). Primary cultures of normal glial cells were prepared in the laboratory by enzymatic dissociation of whole brains from 17-day-old rat fetuses and were maintained in modified Eagle's high-glucose medium containing 10% FBS. Cell cultures were exposed either in 48-well plates, with one stack of two plates placed on the TEM septum, or 60-mm culture dishes, with one group of three stacks of three dishes each placed on the septum and another three-stack group on the bottom of the TEM cell.

As in the Ivaschuk et al. (1997) study, dosimetry considerations were based on Burkhardt et al. (1996). For a nominal slot-average input power of 0.51 W (slot power density 0.9 mW/cm^2), the average SAR for 48-well plates was 5.9 ± 2.8 (SD) W/g, with single-plate minimum and maximum values of 4.1 and 8.1 μW/g. For 60-mm culture dishes, the average SAR was 1.5 ± 1.0 μW/g, with single-dish minimum and maximum values of 0.3 and 2.5 μW/g. The authors indicated that no culture-temperature rises were detected with a thermometer of 0.002°C differential sensitivity for exposures at any of the three input levels.

In the experiments with C_6 glioma cells, cultures in log-phase were incubated for 24 hours. Tritiated thymidine (TdR) was incorporated during the last six hours of that period. The cells were enzymatically dissociated with a serum consisting of trypsin and disodium edetate, and a salt of ethylene-diamine-tetraacetic acid (EDTA) in a calcium-free and magnesium-free saline at specific concentrations of sodium chloride, potassium chloride, sodium bicarbonate, and glucose. The dissociated cells were washed with serum-free medium supplemented with 0.1% bovine serum albumin (BSA) and were counted with a hemocytometer. The cell concentration was adjusted to 80,000 cells/ml in serum-free medium. Cells in 0.5 ml of final volume were plated with complete medium into a 48-well culture plate (six wells for each condition) or were incubated for 18 hours at 37°C before serum was added. The RFEMF exposures were started 18-24 hours after transfer..

Radioactivity perceptible to trichloroacetic acid (TCA) was determined by swabbing the wells with cotton-tipped applicators moistened with 12.5% TCA, and the radioactivities of the swabs were measured in disintegrations per minute (dpm) by liquid-scintillation counting. The average dpm of sham-exposed cultures was 3100-3900 per 40,000 cells, with a maximum variability of 18% and up to a twofold variation between assays.

The results for thymidine incorporation are shown in Table 37 (adapted from Table 2 of the paper) in terms of the mean ratio (E/S) and SE of the counts after 24 hours of exposure at 0.59, 5.9, or 59 μW/g to the counts for sham exposure. Also shown are the E/S and SE after 4 hours of exposure at 5.9 μW/g.

TABLE 37: THYMIDINE INCORPORATION INTO THE DNA OF C6 CELLS AFTER EXPOSURE TO TDMA-MODULATED RFEMF
[Stagg et al. (1997)]

SAR; DURATION	CONDITION	E/S (%) ± SE	NO. OF ASSAYS (Total Sample Size)
0.59 μW/g	Sham	100.0 ± 5.1	3 (18)
24 hours	Exposed	98.6 ± 3.6	3 (18)
5.9 μW/g	Sham	100.0 ± 2.	8 (48)
24 hours	Exposed	109.6 ± 3.5	8 (48)
4 hours	Exposed	103.8 ± 3.7	7 (42)
59 μW/g	Sham	100.0 ± 4.	6 (36)
24 hours	Exposed	98.3 ± 3.9	6 (36)

By t-test, the only significant result was for exposure at 5.9 μW/g for 24 hours (P=0.026). That finding was presented in more detail in Figure 1a of the paper, which showed the results for eight independent

assays of E/S at that RFEMF level (n=6 for each assay) with an error bar for each mean. Those mean E/S values ranged from about 80% (20% below the 100% line) to about 140% (40% above that line), with three of the four E/S values above the line tagged as significant. Displayed in Figure 1b was the mean E/S and error bar for the combined data set of RFEMF exposures, and similarly for the combined sham exposure set (n=48 for both). The RFEMF-mean was about 110% and the sham-mean was 100%, and their error bars were non-overlapping, results that yielded the stated P = 0.026.

The results for thymidine incorporation into primary glial cells exposed for 24 hours at 5.9 or 59 μW/g were presented in Table 3 of the paper. No significant differences in mean E/S were found.

To increase the number of glioma cells initiating cell-cycle progression during exposure, the authors serum-starved cell cultures for 18 hours after plating and stimulated them with 1% or 5% fresh FBS immediately before starting 24-hour exposures at 0.59, 5.9, or 59 μW/g. Those results (displayed in Table 4 of the paper) indicated no significant differences among the mean E/S values for the three exposure levels or for either percentage of FBS. Non-significant DNA-synthesis differences were obtained for similarly treated glial cells as well (data not given).

The authors indicated that growth curves of sham-exposed C_6 cells plated in 60-mm culture dishes showed a log-phase growth that lasted for five days, during which the cell numbers increased exponentially with a mean doubling time of 21.9 ± 1.9 hours. The growth patterns for the cultures exposed to the RFEMF at 1.5 or 15 μW/g were nearly the same as for the sham-exposed cultures; the mean cell-doubling time was 22.7 ± 3.2 hours. In those experiments, the growth phase was followed by a plateau phase for the rest of the experiments (about 13 days). The growth curves for glial cells displayed similar rapid growth followed by a plateau phase, with no significant differences in the curves for sham exposure or RFEMF exposure at 1.5 or 15 μW/g.

The authors concluded that the results of their experiments showed that TDMA-modulated 836.55-MHz RFEMF does not act as a cancer-promoting agent.

Although the findings were negative, some aspects of this study are unclear. First, the cell cultures were plated into either 48-well plates or 60-mm culture dishes. Not discussed was why the numbers of assays and their total sample sizes were fewer than the numbers in the 48-well plates or the 60-mm culture dishes, and differed for the three RFEMF levels used. Were the results for some of the wells or those in culture dishes discarded, and if so, why? Second, why were the mean E/S values for the sham exposures all 1.00, but with SEs that differed from one another? These points, and particularly the absence of the E-data and S-data from which the E/S values and their SEs were derived, would tend to diminish the degree of validity of the negative findings.

Cain et al. (1997) investigated whether TDMA-modulated 836.55-MHz RFEMF and the chemical tumor promoter TPA (12-O-tetradecanoylphorbol-13-acetate) are copromoters that can enhance unregulated growth and focus formation *in vitro* of murine C3H/10T1/2 fibroblasts in coculture with parental C3H/10T1/2 cells. Their methodology was based on techniques developed by Herschman and Brankow (1986, 1987) for determining focus formation by mutant cells UV-TDT10e (10e) in coculture with parental 10T1/2 cell fibroblasts. The authors noted that parental 10T1/2 cells suppress growth of the transformed phenotype of their daughter 10e cells by preventing 10e cells from growing in multilayered foci, which they do when 10e cells grow alone, and that TPA relieves the suppression imposed by the 10T1/2 cells and thereby promotes focus formation.

In this study, 10T1/2 and 10e cells in Eagle's basal medium (BME) and 10% heat-inactivated fetal bovine serum (FBS) were grown respectively in T-75 and T-25 tissue culture flasks at 37 ± 0.2°C within a double-stacked incubator with a continuously flowing mixture of 95% air and 5% CO_2. After 3-4 days in culture, 70%-80% confluent 10T1/2 and 10e cells were seeded into 60-mm petri dishes as cocultures of 1600 10T1/2 cells and 400 10e cells and as the same cell-percentage cocultures plus TPA. Cocultures in 60-mm petri dishes were RFEMF-exposed or sham-exposed in TEM cells like those used in the Ivaschuk et al. (1997) study, for on-periods of 20 minutes alternating with 20-minute off-periods until experiment end (day 29). The cocultures were treated with TPA on days 2, 9, 16, and 23, and the medium was

changed on days 8, 15, and 22. Up to 32 dishes were placed in each TEM cell in stacks of three or four, both on the septum and the bottom of the cell. Dosimetry considerations were based on Burkhardt et al. (1996). For a nominal slot-average input power of 0.51 W (input power density 0.9 mW/cm^2), the mean SAR for 5 stacks of 4 dishes each during the on-periods was 1.5 ± 1.1 (SD) μW/g, with a single-dish minimum of 0.2 μW/g and maximum of 3.0 μW/g.

On day 29, the cells were stained with 1% crystal violet, a cationic triphenylmethane dye that binds DNA, used by Herschman and Brankow (1986), and the stained preparations were video-scanned without operator knowledge of their specific treatment. Focus formation was quantified with video-analysis software. An appropriate optical-density threshold was chosen, based on dark and densely packed foci of 10e cells only. The minimum focus area was set at 0.1 mm^2.

Presented in the text and plotted in Figure 1 of the paper were the mean number of foci per dish, the mean areas of the foci, and the mean density of the foci, all with SEs and all for TPA doses of 10, 30, and 50 ng/ml. Those results were derived from the pooled data for 10 independent experiments with 34, 46, and 42 sham-exposed, and 30, 40, and 43 RFEMF-exposed cultures. At the three TPA doses in increasing order, the mean numbers of foci per dish were 10.1 ± 1.2, 28.3 ± 2.8, and 32.8 ± 2.8 for the sham-exposed cultures and 9.8 ± 1.1, 27.3 ± 3.2, and 35.6 ± 3.5 for the cultures RFEMF-exposed at 1.5 μW/g. Thus, in each case the mean number of foci increased linearly with TPA dose, and at corresponding TPA doses, the differences between the sham-exposed and RFEMF-exposed cultures were not significant. Mean foci area and mean foci density also increased linearly with TPA dose with no significant differences between the RFEMF-exposed and sham-exposed cultures.

Whether a tenfold higher RFEMF power density (SAR: 15 μW/g) would affect TPA-induced focus formation was tested. The pooled results for the mean numbers of foci per dish from four independent experiments, presented in the text and in Figure 2 of the paper, showed smaller and nonlinear increases with TPA dose. At the three TPA doses, the mean numbers of foci per dish were 18.2 ± 3.4, 23.3 ± 4.4, and 30.8 ± 3.7 for the sham-exposed cultures, and 18.4 ± 3.0, 20.6 ± 2.5, and 27.9 ± 3.1 for the RFEMF-exposed cultures. Again, however, there were no significant differences between the RFEMF-exposed and sham-exposed cultures at each TPA dose.

The mean foci area and mean foci density at 15 μW/g, shown only graphically (in Figure 2), also did not indicate a linear rise with TPA dose. With TPA doses of 10 and 30 ng/ml, the mean foci areas were essentially the same, about 25 mm^2 for both the sham-exposed and RFEMF-exposed cultures. At 50 ng/ml, the means for the sham-exposed and RFEMF-exposed cultures were higher than at the two lower doses, and they differed slightly but nonsignificantly from one another. The results for mean-foci density were similar.

In two independent experiments at 0.15 μW/g (the lowest level used), the mean numbers of foci per dish versus increasing TPA dose were 10.1 ± 1.9, 10.6 ± 1.1, and were 13.6 ± 2.0 for sham-exposed cultures. For RFEMF-exposed cultures, they were 5.7 ± 1.1, 10.6 ± 1.2, and 9.13 ± 1.9, results that were also shown graphically in Figure 3 of the paper. At 30 ng/ml of TPA, the means were the same, but from the non-overlapping SE bars in Figure 3 at the other two TPA doses, it would appear that there were significant differences between the means for the sham-exposed and RFEMF-exposed cultures.

However, the authors noted that in the experiments with 0.15 μW/g, the numbers of foci per dish for the sham-exposed cultures ranged only from 10.1 to 13.6, whereas for the sham-exposed cultures in the experiments with 1.5 and 15 μW/g, they ranged from 10 to 35 per dish. The higher mean plating efficiencies for 10T1/2 cells in the experiments with the latter power densities (respectively 32.9% ± 1.7% and 31.8% ± 1.5%) than in those with 0.15 μW/g (27.0% ± 4.0%) led the authors to suggest that exposure to the higher levels may have inhibited the ability of those cells to develop foci in response to TPA.

As did Ivaschuk et al. (1997), the authors remarked that their negative findings are in contrast with their earlier positive results [Cain et al. (1993)] for the same endpoints, using a 60-Hz magnetic field.

The overall conclusion from this and the two companion studies is that exposures *in vitro* of the specific cell types to the TDMA-modulated 836.55-MHz RFEMF neither initiates nor promotes or co-promotes (with TPA) tumor growth. A few questions are raised about some aspects, such as those for Stagg et al. (1997), but considering the studies collectively, the validity of this conclusion is high.

Malyapa et al. (1997a), in a pair of studies, sought to determine whether exposure to 2.45-GHz RFEMF of cultured mammalian cells *in vitro* would cause DNA damage. The cell types selected were the human glioblastoma U87MG and the mouse C3H 10T1/2. The authors used the alkaline comet assay method of Olive et al. (1992) because of its simplicity, low cost, and reproducibility of results.

The exposure facility was a set of broadband radial transmission line systems (RTLs) developed at Washington University for exposing large numbers of culture flasks concurrently, as described in Moros et al. (1998), Pickard et al. (1998), and Straube et al. (1998), all in preparation. Each RTL consisted of a pair of parallel plates driven at the center by a conical antenna, and terminated radially with microwave absorbing layers backed by a lamina of perforated aluminum. The bottom and top plates of each RTL are of aluminum and a metal-faced composite, respectively, with the tops hinged to provide easy access to the flasks.

Up to 10 RTLs were housed on sliding shelves within a warm room. Each RTL could hold 16 T-75 flasks (totaling about 1200 cm^2 of culture area) at a radial distance of 292 mm from the antenna. In this configuration, the fields propagate radially outward, with the electric component perpendicular to the cell-layer plane; the cells are also exposed to the tangential magnetic field perpendicular to both the electric field and the propagation direction.

Monitored continuously were the temperature of the warm room and of the culture medium inside the flasks. To counter the significant heat produced by the RTLs and the instrumentation, cool air was forced into the room and the warm air was exhausted therefrom, thereby holding the room temperature at 35.0 ± 0.25 °C and culture temperatures within all RTLs at 37.0 ± 0.30 °C. Due to the large thermal diffusibility of the aluminum bottom plate, it served as an efficient thermal homogenizer that held all flasks in any RTL within ± 0.30 °C. The microwave energy not taken up by the culture media was absorbed by annular microwave-absorbent material, and the heat generated in the latter was removed by forcing air into the center of each RTL, past the flasks, and radially outward through the porous absorber and perforated aluminum lamina.

Power-deposition uniformity within the flasks was measured with probes at various sites, and with a thermographic camera. Preliminary dose mapping done in one flask with four fiber-optic probes showed that 40-ml volumes of medium (rather than 15 ml) were needed to ensure adequately-high and relatively-uniform SAR. Measurements were made in all of the RTLs to determine the relationship between the temperatures of the aluminum plate and the medium. Fine mapping of SAR was done with a Bowman probe at 18 points in over 64 measurements.

The authors indicated that because of the high thermal diffusion rates, the standard temperature-rise techniques for measuring SARs were not suitable. Based on thermal models, SARs near the bottom of a flask determined from such measurements could be significantly overestimated unless the measurements were done in less than 5 seconds. They therefore utilized a fast temperature-differential technique, cited in Michaelson and Elson (1996). The results with this method showed that an RTL fully loaded with 16 flasks (each holding 40 ml of culture medium) inserted with their long axes parallel to the electric field and equidistant from the antenna, produced a mean normalized SAR near the bottom of the flasks of 89 ± 46 (SD) mW/kg [0.089 ± 0.046 W/kg per mW/cm^2] incident at the proximal end of each flask. At the two nominal input power levels used in the biological experiments (4.8 and 12.3 W), the SARs were estimated to be 0.75 ± 0.39 and 1.93 ± 0.99 W/kg.

Human glioblastoma U87MG and mouse C3H 10T1/2 cells, grown as monolayer cultures in appropriate media, were maintained at 37°C within a humidified incubator having a mixture of 5% CO_2 and 95% air. Only cells in exponential growth phase were used in the experiments. Cell cultures were plated in T-75 flasks (300,000 cells per flask) containing 40 ml of whole medium 48 hours before

exposure. The flasks were gassed with the CO_2 and air mixture and capped tightly before insertion in the RTLs.

As a positive control, cells were irradiated at melting-ice temperature with gamma rays from a ^{137}Cs source calibrated in a small ionization chamber, using a standard traceable to the National Institute of Standards and Technology. To demonstrate the sensitivity of the comet assay for detecting DNA damage, cells were irradiated at the low-dose rate of 6.55 cGy/min. In addition, included in every RFEMF exposure experiment was a sample exposed only to 2 Gy of gamma rays at 1.1 Gy/min.

Both types of cell cultures were exposed in the RTLs to 2.45-GHz RFEMF at 0.7 W/kg for 2, 4, or 24 hours. Other cultures were sham-exposed in the RTLs with no power applied. The cell growth rates were found to be essentially the same for those treatments, and their viabilities exceeded 98%. Immediately after exposure, the cultures were cooled to melting-ice temperature and held there until processed for the comet assays. The flasks were coded and the assays were done without knowledge of the treatment given. Preparation for the comet assay consisted of the following, with duplicate slides made from each preparation:

Cells were suspended in cold phosphate-buffered saline at a concentration of 30-40 thousand cells per milliliter.

Cell suspension samples (0.5 ml) were mixed with 1.5 ml of 1% low-gelling-temperature agarose in phosphate-buffered saline at 42°C. The mixtures were layered immediately onto microscope slides, and each was covered with a glass cover slip. The slides were kept on ice for 1 minute for the agarose to gel.

Immediately after agarose gelling, the cover slips were removed and the slides were placed into cold lysis buffer within an airtight container and allowed to lyse at 4°C overnight in the dark. (Preliminary experiments with positive and negative controls indicated that duration of the lysis treatment for 1 hour (at least) to 24 hours did not affect the pH of the buffer or the outcome of the comet assay.)

The next day, the slides were rinsed three times in a solution designed to remove the NaCl (which is essential for lysis but would reduce DNA migration after lysis). The slides were then transferred to a modified Hoeffler electrophoresis unit having two anode electrodes on one side and two cathode electrodes on the other side. Electrophoresis buffer was added to raise the buffer level to about 5 mm above the agarose.

Electrophoresis was done at 0.6 V/cm for 25 minutes with the buffer kept chilled at 14°C; the electric current was about 40 mA or lower. After electrophoresis, the slides were rinsed in Milli-Q water and stained for 15 minutes with 2.5 μg/ml of propidium iodide dissolved in 0.1-M NaCl.

The treated specimens were excited with 520-540 nm from a mercury lamp and the images of the comets were visualized with an inverted fluorescence microscope. For each experimental point, between 75 and 100 images from the duplicate slides were grabbed in the 24-bit mode with a cooled color CCD camera set to avoid intensity saturation in the comet head, and each image was digitized, and stored. Each image was then analyzed for "comet length" and "normalized comet moment" [endpoints based on Kent et al. (1995)], using the OPTIMAS image analysis software and comet macro program. Threshold settings were selected manually for each comet image to include all pixels above the background. The rest of each image analysis was done by computer without further human input.

The comet length [CL] was measured from the edge of the migrated DNA farthest from the comet head (distal edge) to the edge of the tail farthest from the head in the opposite direction (proximal edge) along the line through the centroids of the head and tail.

For determining the normalized comet moment [NCM], the centroid of fluorescence intensity in the head was taken as zero distance. To derive the DNA content, the fluorescence intensity was then integrated across the width of the head and at 200 regular intervals along the comet length. The result for

each interval was multiplied by that interval's distance from the intensity centroid, and the sum of those values was divided by the total amount of DNA to obtain the NCM.

The authors indicated that all of the data presented were derived from at least three independent experiments, with the results expressed as the average of the means from those experiments and the SE bars. Student's t-test was used to assess statistical significance between RFEMR-exposed and sham-exposed groups.

In the gamma-irradiation experiments to determine the sensitivity of the comet assay in detecting DNA damage, there was no evidence of DNA migration in non-irradiated cells. Also, after exposure at 0.3 cGy, the NCM and CL were not significantly different from their control values. At 0.6 cGY, however, the mean values of the NCM differed significantly from control values for both U87MG cells (P=0.02) and C3H 10T1/2 cells (P=0.03). However, the mean values of CL differed significantly (p=0.03) only for the C3H 10T1/2 cells (with P=0.06 for the U87MG cells). In Figure 2 of the paper, frequency distributions of NCM and CL intensity were shown as bar graphs of the counts per minute at each NCM and CL value for gamma-ray doses of 0, 0.3, 0.6, 1, 3, and 5cGy. The set of NCM bars at each of the successively higher doses indicated a monotonic increase with dose. The results for the CL were similar, but not as pronounced.

Displayed in Figure 3 of the paper were plots of NCM and CL (with SE bars) for U87MG cells after exposure to RFEMF at 0.7 W/kg for durations of 0, 2, 4, or 24 hours and comet assay. No significant differences (P>0.7) were observed in either endpoint with exposure duration, whereas evident were the increases of the NCM and CL with gamma-ray dose up to 5 cGy, plotted on the same graphs. The frequency distributions of the NCM and CL with duration, displayed in Figure 5, also showed no increases with duration. Similar results were shown for C3H 10T1/2 cells in Figures 4 and 6.

Mouse C3H 10T1/2 cells were also exposed at 1.9 W/kg for the same durations, followed by the comet assay, the results of which were displayed in Figure 8 of the paper. Again, there was no evidence of any DNA damage relative to sham exposure, as determined from the measurements of the NCM and CL, in contrast with the positive results from gamma irradiation. The frequency distributions displayed in Figures 9 and 10 support these findings.

The authors noted In their discussion that their gamma-ray dose responses were consistent with the dose-response relationships found by others. They remarked that their negative findings with RFEMF differed from the positive findings of Lai and Singh (1995, 1996). They suggested that the findings of the latter in brain cells of mice exposed to RFEMF *in vivo* may be due to an indirect effect that requires some time to develop, such as 4 hours later. They also indicated that the Olive et al. (1992) method used in their assays was as sensitive as the method used by Lai and Singh.

Malyapa et al. (1997b) performed a similar study to determine whether DNA damage would occur in cultures of mouse C3H 10T1/2 fibroblasts or in human glioblastoma U87MG cells, but from exposure *in vitro* to frequency-modulated continuous-wave (FMCW) RFEMF. The FMCW RFEMF was centered on carrier frequency 835.62 MHz or code-division multiple-access (CDMA) RFEMF with carrier frequency 847.74-MHz, both used for communications by cellular telephones. The exposure system was a set of radial transmission lines (RTLs) like those used in their study with 2.45-GHz RFEMF, but with electromagnetic shielding to avoid interference with local cellular communications.

For the reasons stated in the companion paper, the authors used a fast temperature-differential measurement technique to determine SARs within the cell preparations. The mean normalized SAR for a fully loaded RTL holding 16 T-75 flasks, each containing 40 ml of culture medium was 75 ± 3.8 (SD) mW/kg per mW/cm^2 of incident power density at the proximal end of the flask. The SAR during the biological experiments was estimated to be 0.6 ± 0.3 W/kg for both FMCW-modulated and CDMA-modulated RFEMF.

Both types of cells in the exponential growth phase were investigated in the experiments, but C3H 10T1/2 cells in the plateau phase were also studied. The cells were grown as monolayer cultures in

appropriate media 48 hours before exposure, processed as described in the companion paper, and examined for DNA damage by the alkaline comet assay. Preliminary experiments on rates of cell growth, determined at 2-hour intervals 24 hours before exposure and during 24 hours of exposure to both forms of modulated RFEMF at 0.6 W/kg, yielded the same rates as those for sham exposure. Cultures were also irradiated with gamma rays as a positive control, as described in the companion paper.

Exponentially growing U87MG cells were exposed concurrently in separate RTLs to the two forms of modulated RFEMF at 0.6 W/kg for 2, 4, or 24 hours. One flask was sham-exposed for 4 hours as a negative control, and in some experiments, cell cultures kept in an incubator served as additional negative controls. After treatment, the cultures were subjected to the alkaline comet assay, with the normalized comet moment (NCM) and the comet length (CL) as the parameters (defined in the companion paper) for assessing any DNA damage. The results, presented in Figure 1 of the paper, showed no significant differences ($P>0.7$ by Student's t-test) in NCMs or CLs for exposure to modulated RFEMF of either type versus exposure duration. The bar-graph frequency distributions (displayed in Figure 2) also showed no increases in either parameter, but the results for the gamma-ray exposures (also displayed in Figure 1) were again monotonic increases in both parameters with dose (up to 5 cGy).

Negative results were also obtained for C3H 10T1/2 cells in exponential growth phase (Figure 3): no significant differences ($P>0.8$) in NCMs or CLs for exposure to either type of modulated RFEMF versus sham exposure.

In another experiment said by the authors to better simulate *in vivo* conditions in which most of the cells are nonproliferating, C3H 10T1/2 cells in the G_0 plateau phase were similarly exposed for 2, 4, or 24 hours to RFEMF of either modulation type or sham-exposed. Again, no significant differences ($P>0.7$) in NCMs or CLs were observed. In addition, exponentially-growing and plateau-phase C3H 10T1/2 cells were both RFEMF-exposed or sham-exposed for 2 hours, but then were incubated for 4 hours at 37°C before being processed for comet assay. The authors stated that this experiment was done to model, under *in vitro* conditions, the reported results of Lai and Singh (1996) on DNA damage in brain cells of rats from exposure *in vivo* to 2.45 GHz RFEMF with 4-hour post-exposure cell processing and use of the comet assay. Once more, no significant differences in NCMs or CLs for the cells in either phase were seen (Figures 6-9 of the paper).

Separate experiments were done to determine whether labeling C3H 10T1/2 cells in growth phase with 5-bromo-2'-deoxyuridine (BrdU) would enhance any DNA damage detectable by the comet assay after RFEMF exposure. The authors noted that this halogenated pyrimidine is known to sensitize proliferating cells toward DNA damage from X-rays or ultraviolet light. The results for RFEMF exposure showed no changes in growth kinetics or cell viability for cells processed in near-dark conditions. Because fluorescent light does not cause significant DNA damage in unlabeled cells, but can do so in cells labeled with BrdU, a cell culture within one T-75 flask was BrdU-labeled, exposed to a 30-W fluorescent-light source at 50 cm distance for 1 hour at 37°C, and processed for the comet assay. This positive-control experiment yielded significant increases ($P<0.04$) in NCM and CL (Figure 13).

Malyapa et al. (1997a), in the studies found no experimental evidence for DNA damage from exposure *in vitro* of cultures of human glioblastoma U87MG and mouse C3H 10T1/2 cells to 2.45-GHz RFEMF at SARs 0.6 or 1.9 W/kg. Those findings are contrary to the positive findings reported by Lai and Singh (1995, 1996) for cultures of brain cells from mice exposed *in vivo* to the same frequency. However, the RFEMF-dosimetric aspects and comet-assay methodology in the Malyapa et al. (1997a) study were far more refined than the estimates of mouse-brain SARs and the comet-assay methodology used by Lai and Singh. In addition, the use of cell cultures exposed to gamma-rays as a positive-control by Malyapa et al. (1997a) reinforce the confidence in their experimental data. Thus, no scientific credence can be given to the positive findings of Lai and Singh (1995, 1996).

As noted earlier about the two Lai and Singh studies, it seems likely that significant brain autolysis may have occurred during the period between mice euthanization after exposure end and the start of cell-culture preparation for assay. Thus, it is unclear whether such studies involving RFEMF exposure *in vivo* could be done without introducing non-RFEMF-related factors.

The negative findings of Malyapa et al. (1997a, 1997b) on postulated DNA damage from exposure of cultures of human glioblastoma U87MG and mouse C3H 10T1/2 cells to FMCW-modulated 835.62 MHz or to CDMA-modulated 847.74 MHz RFEMF (frequencies used for cellular-telephone communications) at 0.6 W/kg SAR do not support reports, in some epidemiologic studies, of cancer incidence due to exposure to the RFEMF emitted by cellular telephones or their base stations.

4.3 SUMMARY OF RFEMF AND CANCER IN NON-HUMAN MAMMALS

The findings on cancer induction and promotion in mammals from RFEMF exposure *in vivo* are summarized in Table 38 (A through I), and those for exposure *in vitro* in Table 39 (A through G).

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Prausnitz and Susskind (1962)	Sought were effects of high-level pulsed RFEMF in mice.	Exposed were 20 groups of 10 mice to 2-s, 9.3-GHz pulses, 500 pps, at 100 mW/cm^2 average power density, for 4.5 minutes a day, 5 days a week, for 59 weeks. The subsequently estimated SAR was about 45 W/kg. The controls were 100 sham-exposed mice.	The body-temperature rise that caused death in half the animals (LD50) was 6.7°C, attained in 12 minutes, so the exposures for 4.5 minutes were sublethal. However, the death rate was greater in sham-exposed than in RFEMF-exposed mice: On exposure completion, 50% of the control mice and 65% of the RFEMF-exposed mice were still alive. The deaths in both groups were attributed to a pneumonia infection introduced accidentally. At necropsy, liver abcesses were found in both groups, but could not be quantified because of tissue loss by autolysis. Also found was leukosis in the exposed and control groups, with higher incidence in the RFEMF-exposed mice. The authors described leukosis as a "cancer of the white blood cells", but leukosis is defined as an abnormal rise in the number of circulating white blood cells whatever the cause, rather than as a form of cancer.	Various factors can give rise to leukosis, including stress, endocrine-system disturbances and infections such as the pneumonia noted, which may have caused the observed liver abscesses. Two other points need be made: 1) Leukosis incidence was higher in the RFEMF-exposed mice, but so was their survival percentage, an unusual finding for most forms of leukemia. 2) Leukosis incidence was higher during but not after completion of the RFEMF-exposure series, implying a spontaneous remission of the "cancer", an. improbable outcome. This study did not yield any valid positive or negative evidence that exposure of animals to high RFEMF levels induces cancer, a conclusion supported by reanalysis of the data by Roberts and Michaelson (1983).
Skidmore and Baum (1974)	Investigated was whether rapid changes in the pulsed electric and magnetic fields from EMP (the RFEMF with the broad frequency spectrum from a nuclear-bomb burst) would induce injuries in biological systems with high cell-turnover rates.	Both male and female rats and male mice were exposed almost continuously to electromagnetic pulses from an EMP simulator for 38 weeks, for a total of 100 million pulses at 5 pulses per second. Each pulse had a rise time of 5 ns, a fall time of 550 ns, and a peak electric field of 447 kV/m.	Various endpoints were examined, which yielded significant differences in a few blood parameters between exposed and control groups, but with the possibility of the presence of uncontrolled non-RFEMF factors. More relevant to this report, 20 female rats were continuously exposed to the EMP RFEMF, with 20 rats as controls, and were observed for possible development of mammary tumors. At age 1 year (following exposure to 10 million pulses), no mammary tumors were found in either group. Also exposed to the EMP were 50 male AKR/J mice, a strain prone to spontaneous development of leukemia between 6 and 12 months of age, with 50 mice as controls. After 33 weeks (86 million pulses), the 42 surviving EMF-exposed mice and 24 control mice were examined for the presence of leukemia. Nine of the exposed mice (21%) and 11 of the control mice (46%) were leukemic, indicating that the exposure to EMP was not involved in leukemia development.	The findings of no mammary tumors or leukemia appear to be sound. Because of the short duration and shape of the EMP, its broad frequency spectrum, and low pulse-repetition rate, efforts to determine the equivalent free-space average power density and/or the SAR for comparison with the levels in other studies seemed inappropriate.

TABLE 38A: CANCER INDUCTION AND PROMOTION IN MAMMALS FROM RFEMF EXPOSURE *IN VIVO*

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
McRee et al. (1981)	Sought was whether RFEMF is mutagenic in mice by analyzing induction of sister chromatid exchange (SCE) after exposure.	Exposure of 12 ten-week-old mice to far-field 2.45-GHz CW RFEMF at 20 mW/cm^2 8 hours a day for 28 days. Most SARs were close to 27 W/kg. The controls consisted of 12 sham-exposed mice and 12 held in home cages.	Immediately after treatment completion, the SCEs in the femur bone marrow were determined, and the mitotic index was scored as the proportion of metaphase cells in a sample of more than 1,000 bone marrow cells from each mouse. The results for the RFEMF, sham, and control groups were comparable, about 3 SCEs per metaphase cell, an indication that the RFEMF did not significantly affect the sister chromatid exchanges. Also, analysis of the mitotic-index values showed that the RFEMF had no significant effect on the rate of proliferation of bone-marrow cells.	The results for only 3 mice of each group were presented; those for the other mice were eliminated primarily because the cell counts per sample were inadequate, a feature inherent in the specific type of assay used.
Saunders et al. (1983)	The authors, noting that Saunders and Kowalczuk (1981) had showed that exposure of the rear halves of mature male mice to 2.45-GHz RFEMF at half-body SARs of 43 W/kg severely reduced sperm production, investigated whether such exposure was also mutagenic for male germ cells.	Four groups each of 6 sexually mature male mice were anesthetized, and their rear halves were exposed to 2.45-GHz RFEMF for 30 minutes at 43 W/kg. For each group, 6 mice were similarly sham-exposed. Rectal temperatures, measured immediately after RFEMF- and sham exposure, were 41.5°C and 34.1°C, respectively.	After treatment, each male was mated with pairs of sexually mature virgin females each week for 8-10 weeks. The females were euthanized 14 days after mating. The percentages of RFEMF- and sham-exposed females rendered pregnant were comparable for the first 2 weeks, diminished for weeks 3 through 8, and became comparable again by mating week 10. For each week, the numbers of corpora lutea per pregnant female of the RFEMF and sham groups were comparable. The numbers of total implants for the RFEMF groups diminished by week 5 to about half that for the sham groups and then recovered by week 10. For each week, the percentages of live implants to total implants for the RFEMF and sham groups were comparable.	The diminution in total implants was ascribed to decreased male fertility, in consonance with the previous findings of Saunders and Kowalczuk (1981). However, the absence of significant differences in the percentages of live implants to the total implants for the RFEMF and sham groups provided no evidence of an RFEMF-induced dominant lethal mutagenic effect.
Szmigielski et al. (1982)	Investigated were: whether RFEMF exposure would reduce the natural resistance of Balb/c mice to lung cancer cells injected before exposure, increase the incidence of skin cancer in Balb/c mice depilated and painted with the carcinogen BP, or increase breast-tumor incidence in female C3H/HeA mice, known to have a high spontaneous incidence of such tumors.	Groups of 40 Balb/c mice were exposed to 2.45-GHz RFEMF at 5 mW/cm2 or 15 mW/cm2 2 hours a day, 6 days a week, for 1 to 6 months. In addition, Balb/c mice were grown in small compartments for 1 to 8 months to cause chronic stress syndrome. Groups of 40 C3H/HeA mice were similarly exposed and were palpated every 2 weeks for breast tumors.	In the lung-cancer study, the injected Balb/c mice RFEMF-exposed at 5 mW/cm^2 and killed 14 days later yielded 6.1 ± 1.8 neoplastic nodules versus 10.8 ± 2.1 nodules for exposure at 15 mW/cm^2, a significant difference. The result for those raised in confinement and injected with sarcoma cells was 7.7 ± 2.0 nodules, about the same as that for exposure at 5 mW/cm^2. Regarding breast cancer, the cumulative results were expressed as mean cancer-development and survival times. The results for 5 mW/cm^2 and confinement stress were similar and lower than for 15 mW/cm^2. The skin-cancer results were mixed. The values for 5 and 15 mW/cm^2 were comparable, indicating the lack of a clear dose-response relationship.	The authors noted that 2.45-GHz RFEMF is close to resonance for mice and the highest absorption by humans at this frequency would be almost two orders of magnitude lower than for mice. Also, RFEMF absorption by humans at their resonant frequencies (60-70 MHz) would be about 20% of that by mice at their resonant frequencies.Thus, any RFEMF-induced increases in skin-cancer incidence were probably due to the heat stress rather than any postulated intrinsic carcinogenic properties of the RFEMF.

TABLE 38B: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Santini et al. (1988) [Brief communication]	Investigated was whether low-level RFEMF exposure of black mice (strain C57BL/6J) would have any effect on the development of B16 melanoma or the survival times of the mice.	Three groups of 15 5-week-old female mice each were exposed to 2.45-GHz RFEMF at 1 mW/cm^2 (SAR 1.2 W/kg). One was exposed to CW RFEMF and another to pulsed RFEMF, both at the same average power density, for 2.5 hours a day in 6 sessions a week until death (up to 690 hours total). The third group was sham-exposed. The pulsed RFEMF consisted of 10-ms bursts of 10-s spikes, 5 seconds between spikes, and 30 ms between bursts.	After 15 days of RFEMF exposure, all 3 groups were implanted with 3 million (0.1ml) melanoma cells. RFEMF- and sham exposures were continued after inoculation until all of the mice died. Tumor sizes were measured at periodic intervals. The results were displayed as bar graphs of mean tumor surface areas and standard deviations at time intervals of 9, 12, 16, 20, 23, and 27 days after inoculation. The mean tumor area for each group increased with time, as expected. At each time interval, the SD bars for the three groups overlapped; the authors stated that there were no significant differences among the groups, but did not show any experimental data. Mean survival times and SDs for the mice afflicted with B16 melanomas were tabulated. The results for the sham, CW, and pulsed groups respectively were 25.9 ± 6.2, 24.4 ± 7.9, and 26.6 ± 8.5 days. The differences were not significant.	Although the findings of this study showed no significant differences among the RFEMR-exposed and sham-exposed mice in melanoma development and survival times, the credibility of those negative findings is vitiated to some degree by the absence of experimental data, presumably because the paper was a brief communication.
Chou et al. (1992)	Investigated were 155 endpoints in a group of 100 Sprague-Dawley rats exposed concurrently to circularly polarized 2.45-GHz RFEMF throughout their lifetimes (up to 25 months) with another group of sham-exposed rats.	The exposures were done under specific-pathogen-free (SPF) conditions in individual cylindrical waveguides at an average power density of about 0.5 mW/cm^2, with whole-body SARs in the range 0.4 W/kg for a 0.2-kg rat to 0.15 W/kg for a 0.8-kg rat.	For most of the 155 endpoints, no significant differences were found between the RFEMF-exposed and sham-exposed rats. One general finding was that the median survival time of the RFEMF group was slightly longer than for the sham group (688 versus 663 days). Relevant were incidences of lesions noted as non-neoplastic or neoplastic, with chronic glomerulonephropathy (non-neoplastic) as the most frequent cause of death, but found in fewer of the RFEMF-exposed than the sham-exposed rats. Primary malignancies were found in 18 RFEMF-exposed rats but only in 5 sham-exposed rats. The difference in incidence of each specific type of malignancy between the RFEMF and sham rats was nonsignificant, but the collective incidences (totals of 18 versus 5 rats) without regard to the site or organ of occurrence differed significantly.	This paper is a recent summary of a group of technical reports cited in the reference list. The difference in malignancy incidence, 18 RFEMF-exposed versus 5 sham-exposed rats was statistically significant, but its biological significance is open to question: This difference was found by collapsing sparse data without regard to the specific type of malignancy or tissue of origin. Also, the incidence of the malignancies in the RFEMF-exposed rats was similar to those of untreated control rats of the same strain maintained under similar SPF conditions, an indication that the difference was not likely due to the RFEMF.

TABLE 38C: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Salford et al. (1993)	The authors, noting that the rat glioma cell line RG2 causes glioma-like tumors in Fisher 344 rats when injected into their brains, investigated whether RFEMF exposure after such treatment would promote the growth of such tumors.	Seventy-four Fisher 344 rats were injected with RG2 cells into the head of the right caudate nucleus. Starting on day 5 after injection, 37 of the rats were exposed for 7 hours a day, 5 days a week for 2 or 3 weeks, to 915-MHz CW RFEMF or to 0.57-ms or 6-ms pulses of the RFEMF at several pulse-repetition rates. The SARs were from 0.0077 to 0.4 W/kg for 0.57-ms pulses and 1.0 to 1.67 W/kg for 6-ms pulses. Each rat was matched with a sham-exposed control.	All of the rats developed tumors. Histopathologic examination showed the tumors usually to be solid with minor necrotic areas not correlated with treatment, tumor size, or time elapse from inoculation to death. There were no signs of brain damage outside the tumor areas, either necrosis, gliosis, or inflammatory changes ascribable to RFEMF exposure. Measurements of tumor area were expressed in terms of the tumor area of each RFEMF-exposed rat (E) and that of its matched control (C), and the results were tabulated as relative changes (in percent). Most of the changes well exceeded 100%, but from using Student's t-test on paired values, the authors concluded that they had found no significant differences between the RFEMF-exposed and the control rats. Nevertheless, they remarked that they had found some RFEMF-exposed rats with much larger tumors than their controls.	Because of the large variabilities in the data for both the RFEMF-exposed and the matched control rats, the results of this study do not constitute scientifically credible evidence that the RFEMF did or did not alter tumor growth initiated by injection of rat glioma cells from cell line RG2 into the brains of Fisher 344 rats.
Lai and Singh (1995)	In this study, the authors investigated whether acute exposure of rats to 2.45-GHz pulsed or CW RFEMF would affect the DNA in brain cells.	Rats were exposed for 2 hours to 2.45-GHz pulsed RFEMF at 1 or 2 mW/cm^2 average power density; the whole-body SARs were 0.6 or 1.2 W/kg. At 0.6 W/kg, the local SARs in 8 brain regions were in the range 0.5-2.0 W/kg. Immediately after exposure or 4 hours later, the rats were euthanized and their hippocampi and the rest of their brains were assayed separately for single-strand DNA breaks. In a second experiment, rats were exposed for 2 hours to 2.45-GHz CW RFEMF at 2 mW/cm^2, euthanized immediately or 4 hours later, and their whole brains assayed.	The assays were done by the method of Singh et al. (1994a): After brain excision, samples were treated to reveal any single-strand DNA breaks in microgels on slides for alkaline electrophoresis, a process for spatially spreading DNA fragments by their molecular weights. The slides were stained with a fluorescent dye, and 50 cells on each slide were assayed by fluorescence microscopy. No numerical data were presented; instead, displayed were bar graphs of mean migration length [MML] of DNA as an index of single-strand breaks. In the first experiment, the assays of the hippocampi and the rest of the brains done immediately after exposure showed no statistically significant effect at either RFEMF level. The MMLs for the assays 4 hours post-exposure at either level significantly differed from those for sham exposure, but the difference between the MMLs for the two RFEMF levls was not significant. The whole-brain assays done both immediately and 4 hours post-exposure in the second experiment showed significant differences in MMLs between RFEMF and sham exposure.	Among the questionable aspects are: 1) Dosimetry measurements were not performed. Instead, Chou et al. (1985) was cited, but those authors had said that the local SARs in eight brain regions differed widely at a fixed whole-body SAR. Also, not known were the temporal variations of local SARs due to rat movements or changes in orientation or configuration during exposures. 2) The small numbers of rats shown in the bar graphs seem to imply that only one group of rats was treated at each level in each experiment, with immediate or 4-hour delays in euthanizing the rats. If so, the positive findings, if real, were not statistically robust. 3) Since only 50 cells on each slide were assayed, to what extent did the remaining cells differ? 4) Was autolysis an uncontrolled factor?

TABLE 38D: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Williams (1996) [Comment on Lai and Singh (1995)]	See Lai and Singh (1995)	See Lai and Singh (1995)	Williams commented that the reported absence of an increase of single-strand DNA breaks (SSDB) assayed right after pulsed-RFEMF exposure and a significant increase of SSDB assayed 4-hours later is not consistent with the findings of other types of radiation-induced SSDB. In the latter, breakage is generally highest at exposure end or shortly later, after which DNA repair begins to ameliorate damage [three references cited]. Williams also pointed out that the SSDB background from sham exposure in the 2nd experiment was about 2/3 of that in the 1st, a sign of significant artifact in the assay processes. The author aAlso remarked that the reported non-decrease of SSDB after the pulsed RFEMF and 4-hour assay delay may be taken as the absence of repair of the putative DNA breaks, again not consistent with the knowledge about processing DNA damage resulting from other forms of radiation.	Perhaps the most significant point by Williams was the likelihood of the presence of artifact in the assays. If true, then little if any credence can be given to any of the findings of Lai and Singh (1995) irrespective of the other points noted previously.
Lai and Singh (1996b) [Response to Williams (1996)]	See Lai and Singh (1995)	See Lai and Singh (1995)	Lai and Singh (1996b) cited other references to indicate that the extent and duration of the DNA repair processes after damage is not constant but depends on the specifics of the experiments, such as the type of radiation, exposure duration, and details of the assay processes.	This rebuttal, which involved counter-citations regarding DNA damage repair processes, does not address Williams' point about the possible presence of artifact.
Lai and Singh (1996a)	The authors did a similar study with rats, but toward seeking double-strand as well as single-strand DNA breaks in rat brain cells from exposure to RFEMF.	The exposures were to CW and pulsed 2.45-GHz RFEMF for 2 hours, done concurrently with 2 sets of 8 rats each at 1.2 W/kg mean whole body SAR, with 8 sham-exposed rats for each set; also, the rats were euthanized and their brains excised only 4 hours post-exposure.	Processing of the excised brains was similar to that used in Lai and Singh (1995) to obtain slides for alkaline microgel electrophoresis. Two slides each for assaying single-strand and double-strand breaks were prepared from the brain of each rat, and 50 representative cells were scored for each slide. Single-strand DNA breaks for both forms of RFEMF were comparable to each other but were significantly larger than those for sham exposure; however, the bar graphs of the mean distributions of migration length (in 10-micron bins) displayed rather subtle differences. The results for double-strand breaks were similar to those for single-strand breaks but less pronounced.	As in the Lai and Singh (1995) paper, the lack of meaningful dosimetry and the unknown time variations thereof due to rat movements and other changes in body geometry are the major criticisms that vitiate any results considered related to RFEMF exposure. In addition, the likelihood is high that autolysis did confound the results. Of interest is that this may not have been a problem in the Singh et al. (1994a) study in vitro of human-lymphocyte DNA exposed to ionizing radiation.

TABLE 38E: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Lai and Singh (1997)	The authors investigated their hypothesis that free radicals were involved in the rat-brain DNA damages assayed 4 hours after in vivo exposure to the 2.45-GHz pulsed RFEMF at 2 mW/cm² (1.2 W/kg).	Rats were exposed to the pulsed RFEMF for 2 hours but were injected with melatonin or spin-trap compound PBN right before and right after exposure, both efficient free-radical scavengers. The authors expected that those substances would block the RFEMF damage found in the previous studies.	Melatonin in ethanol-saline solution (its "vehicle") or PBN in physiological saline (its "vehicle") was injected in rats and the results were compared with RFEMF-exposed rats injected only with their vehicles, and with sham-exposed rats injected with either substance or its vehicle. Also included in each experiment was a group of unhandled rats. Fifty cells on each slide were randomly chosen for scoring, but damaged cells, said to occur equally in all groups, were not measured. Single-strand MMLs for the sham-vehicle group and cage controls were about the same but were much higher for the RFEMF-vehicle group, taken as indicating that the RFEMF had increased the MML. Also, the MMLs for the sham-melatonin and RFEMF-melatonin groups were about equal, taken as confirming that melatonin blocked the effect of the RFEMF by scavenging the free radicals. The double-strand MMLs for melatonin and PBN were qualitatively similar to those for single-strand MMLs. The results for the distributions of cell percentages in 10-micron bins were less clear. For example, the unhandled-control group had a bi-modal cell distribution, whereas the distribution for the sham-vehicle group was within a single narrower range.	During the 2 hours between the two injections of melatonin or PBN, did the rats tend to sleep or otherwise differ in behavior or metabolism from those not so treated? Unclear is to what extent, if any, such damage had occurred on the slides assayed, and whether such damage had influenced the selection of the cells thereon for measurement. Also unclear was why error bars were not shown in some of the bar graphs. As noted previously, the lack of meaningful dosimetry and possible confounding of the results by autolysis are major criticisms. Thus, little if any scientific credence can be given to the findings herein.
Wu et al. (1994)	Investigated was whether colon cancer induced by dimethylhydrazine (DMH) in young mice would be affected by exposure to RFEMF. For comparison, the effects of using DMH with 12-O-tetradecanoylphorbal-13-acetate (TPA) were also studied.	Balb/c mice of both sexes 4-5 weeks of age were divided into groups A: controls, B: DMH only, C: DMH + 2.45-GHz RFEMF, and D: DMH + TPA. RFEMF- and sham exposures of group-C mice were for 3 hours a day, 6 days a week, for 5 months in single cages at 10 mW/cm² (10-12 W/kg).	Once per week for 14 weeks, the mice of groups B, C, and D were injected with DMH in doses of 15 mg/kg, followed during the next 8 weeks with doses of 20 mg/kg. Group-A mice were injected with saline instead of DMH. Starting 3 weeks after first DMH treatment, group-D mice were injected with TPA once a week for 10 weeks, and groups A, B, and C were injected with saline instead of TPA. All mice were euthanized at week 25 and the tumor nodules in the mucosa of the colon were tabulated. None of the control mice developed colon tumors and the differences in numbers of mice with tumors among groups B, C, and D were not significant. Evidently DMH functioned as an initiator in those three groups, but the RFEMF exposure did not augment the effect of DMH (group C) nor did TPA (group D).	The antenna and RFEMF-exposure arrangement were not adequately described. From the description, the RFEMF- and sham exposures were apparently not done concurrently. Also unclear was how the specific numbers of mice in each group were selected. Such points tend to reduce confidence in the negative findings of this study but not fatally so.

TABLE 38F: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Repacholi et al. (1997)	Investigated was whether transgenic mice that carry the E-Pim1 gene and are therefore prone to the spontaneous occurrence of lymphoma would show an increased incidence from exposures to modulated 900-MHz RFEMF such as in Australian digital mobile telecommunications.	Exposed were 101 female mice to modulated 900-MHz RFEMF for two 30-minute periods a day for up to 18 months. Cages with 5 mice each were spaced around a vertical monopole antenna at the center of a ground plane within a small room lined with aluminum sheeting. The levels ranged from 2.6 to 13 W/m^2 (0.26 to 1.3 mW/cm^2). Normalized SARs ranged from 0.003 to 0.31 W/kg per W/m^2. One hundred mice were sham-exposed.	The primary results were tabulated as incidences of: lymphoma characterized as lymphoblastic (LB) or non-lymphoblastic (Non-LB); renal disease only or with both lymphoma and renal disease; other diseases such as deaths from causes including dehydration, injuries, hepatoma, and amyloidosis; and undiagnosable cases (dead mice with tissues too autolysed for analysis). Shown were the following diagnoses: 6 exposed- and 3 control mice with LB lymphoma, 37 exposed- and 19 control mice with Non-LB lymphoma, and 12 exposed- and 8 control mice with other disease. The latter exhibited abnormal clinical signs, but no evidence of lymphoma on necropsy. Mice with renal disease alone or with lymphoma occurred about equally in exposed and control mice. The cumulative probabilities for occurrence of LB- and non-LB lymphoma in the exposed group were both significantly higher than for the sham group, but lacking were details of the calculations.	Engineering aspects were seriously flawed: e.g. the aluminum lining of the exposure-chamber walls created a reverberant microwave cavity that had a large number of complex field modes rather than the stated far-field conditions. Also, interactions among the 5 mice in each cage may have caused large SAR variations. Using the transgenic mice seemed inappropriate, because the sham-exposed mice were also susceptible to lymphoma and were therefore already compromised. The lack of colony-control mice neither RFEMF- nor sham-exposed for comparison with the sham-exposed mice is a basic problem. Thus, very little if any scientific credence can be ascribed to the findings herein.
Toler et al. (1997)	Thsee authors Investigated whether chronic exposure of mammary-tumor-prone mice to low-level 435-MHz RFEMF would promote the earlier onset, faster growth rate, or higher incidence mammary tumors.	Two hundred cancer-prone C3H/HeJ female mice were exposed 22 hours a day, 7 days a week for 21 months to horizontally-polarized, 435-MHz, pulsed RFEMF at 1.0 mW/cm^2 average power density. A level selected to simulate exposure to the RFEMF from the U.S. Air Force's PAVE PAWS surveillance tracking system operating in the band 420-450 MHz. By calorimetry, the SAR was 0.32 W/kg. Sham-exposed were 200 other mice. On 4 days of each week, 50 RFEMF-exposed and 50 sham-exposed mice were palpated for tumors.	At treatment end, the 47 RFEMF-exposed and 47 sham-exposed surviving mice were necropsied and histologically examined. Tabulated were the numbers of mice in both groups with neoplastic and non-neoplastic tumors or lesions at various sites, and a chi^2 test based on overall proportion of tumor-bearing mice. Also estimated was the probability of survival and differences in survival rates between the RFEMF-exposed and sham-exposed mice. A total of 85 adenocarcinomas of the mammary gland appeared in 77 of 193 RFEMF-exposed mice versus 82 adenocarcinomas in 74 of 190 sham-exposed mice, incidences not significantly different between the groups. The authors noted that the morphology of the mammary carcinomas found was consistent with that expected in a high-incidence mouse strain such as one having the mammary tumor virus. The median times for tumor onset in the RFEMF-exposed and sham-exposed mice did not differ significantly.	The authors noted that the findings were consistent with those of Santini et al. (1988) with C57/6J mice, Wu et al. (1994) with BALB/c mice, and Frei et al. (1998) with C3H/HeJ mice, and that the findings support those of Chou et al. (1992) with Sprague-Dawley rats. They also noted that Szmigielski et al. (1982) had found that cancer-prone C3H/HeA mice exposed to 2.45-GHz RFEMF at 15 mW/cm^2 for 2 hours a day, 6 days a week, for several months developed tumors earlier than their sham-exposed controls. Those authors suggested that this response might have been due to chronic stress.

TABLE 38G: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Vijayalaxmi et al. (1997) Vijayalaxmi et al. (1998a, 1998b) [Corrections]	Investigated was the extent of cytogenetic damage to peripheral-blood cells and bone-marrow cells of mammary-tumor-prone C3H/Hej mice from chronic exposure to 2.45-GHz CW RFEMF, using the micronucleus assay.	Exposed to the RFEMF were 100 C3H/Hej mice 20 hours a day, 7 days a week, for 18 months at 1.0 W/kg, with 100 mice sham-exposed and 25 mice as sentinels. On treatment completion, the surviving 62 exposed and 58 sham-exposed mice were killed. Identified in smears of peripheral blood and bone marrow were young polychromic erythrocytes (PCEs), mature erythrocytes, and micronuclei by color with fluorescence microscopy. As positive controls, the 7 surviving sentinels were injected with mitomycin C (MMC) to induce micronuclei formation and similarly processed.	Initially reported were nonsignificant differences in group-mean percentages of PCEs in either peripheral blood or bone marrow among the RFEMF-exposed, sham-exposed, and MMC-treated mice. In addition, the numbers of micronuclei per 2,000 PCEs for the RFEMF-exposed and sham-exposed mice did not differ significantly, but the numbers for those treated with MMC as a positive control were tenfold higher ($p<0.001$) in both peripheral blood and bone marrow than for the RFEMF-exposed and sham-exposed groups. Mammary tumors were found in 12 of the 62 RFEMF-exposed and 8 of the 58 sham-exposed surviving mice. The mean percentages of PCEs in both peripheral blood and bone marrow also did not differ significantly between groups. Noted subsequently in a correction was that the mean micronuclei were correctly given as per 1000 PCEs, but that the SDs had been calculated per 2000 instead of 1000 PCEs as stated in their tables. When correctly calculated, the differences between the two groups were significant but the increase was only 1 micronucleus per 2000 PCEs.	The authors concluded that such chronic exposure to 2.45-GHz RFEMF, the frequency approximating the highest energy absorption rate (resonant frequency for highest whole-body SAR in mice) did not show any genotoxicity. The experimental protocol and dosimetry in this study are highly credible, and the finding of no genotoxicity is reinforced by the positive results obtained with the sentinel mice that were injected with mitomycin C, a known chromosome-breaking agent (clastogen).
Frei et al. (1998)	In this investigation, as in Vijayalaxmi et al. (1997), 100 tumor-prone C3H/HeJ mice were chronically exposed to low-level 2.45-GHz CW RFEMF, but to determine whether such exposure promotes earlier onset, greater incidence, or faster growth rate of mammary tumors.	The exposures were for 20 hours a day, 7 days a week, for 18 months, at a mean whole-body SAR of 0.3 W/kg, to match the level used by Toler et al. (1997) with 435-MHz RFEMF. As before, 100 mice were sham-exposed, and 25 mice served as sentinels. Moribund mice and the mice that had died spontaneously were necropsied; the survivors were necropsied at the end of treatment.	Histopathologic examinations revealed neoplasms in various tissues, with mammary-gland carcinomas most frequent. Single or multiplasms had occurred in 54 sham-exposed and 44 RFEMF-exposed mice, diagnosed previously by palpation in 52 sham-exposed and all 44 RFEMF-exposed mice. The difference between the groups was not significant. Plots of cumulative incidence of mammary tumors versus weeks of treatment were coincident up to about 55 weeks. Beyond that time, both plots showed a trend toward higher incidence in the sham group relative to the RFEMF group, but a survival analysis of time-dependent differences in tumor onset indicated no significant difference between the groups. The neoplasms found in other tissues were tabulated. The only significant difference between the groups was for lung tumors: alveolar-bronchiolar adenoma in 4 of 97 sham-exposed mice and none in 99 RFEMF-exposed mice.	The authors noted that the results of their study are consistent with those of Santini et al. (1988), Wu et al. (1994), and Toler and Shelton (1995) [meeting abstract] who had found that long-term exposure to low-level RFEMF did not promote cancer expression. As with the investigation by Vijayalaxmi et al. (1997), the experimental protocol and the dosimetry are highly credible, as are the findings.

TABLE 38H: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Malyapa et al. (1998a) Malyapa et al. (1998b)	In a study subsequent to their *in vitro* studies, the authors sought possible DNA damage in the brains of rats exposed *in vivo* to 2.45-GHz RFEMF under conditions similar to those used by Lai and Singh (1994).	Sprague-Dawley rats were exposed in pairs, one to 2.45-GHz CW RFEMF for 2 hours at 2 mW/cm^2 and the other sham-exposed. Immediately or 4 hours after exposure, the rats were killed and each rat's and cerebral cortex and hippocampus were processed for the alkaline comet assay by the Olive et al. (1992) method. In separate experiments, the rats were killed together by CO_2 asphyxiation or in sequence by guillotine.	In a first series of experiments using concurrent CO_2 asphyxiation, differences in comet lengths [CLs] were seen between rat pairs that depended on whether the RFEMF-exposed rat was assayed before the sham-exposed rat or conversely. Also, little DNA damage was seen in the rats killed first, but significant damage was seen in those killed second regardless of treatment, implicating the time elapse between exposure and euthanasia. In the next set, one rat of each pair was asphyxiated and its brain excised before the other rat was euthanized, thereby eliminating the time elapse and the DNA-damage difference observed. In the third set, the rats were successively decapitated by guillotine after exposure. Comparison of the results with those for the second set showed no significant differences between RFEMF-exposed and sham-exposed rats in CLs or normalized comet moments [NCMs] regardless of euthanasia method. Also, based on comparisons with results for gamma-ray exposures as a positive control, no significant differences in CLs or NCMs could be ascribed to the RFEMF exposure.	The negative findings of this *in vivo* study of possible DNA damage in the brains of rats from RFEMF exposure, as determined by the alkaline comet assay method of Olive et al. (1997), do not confirm the positive findings reported in the *in vivo* study of Lai and Singh (1994) using the alkaline comet assay method described in their paper.

TABLE 38I: HUMAN RFEMF EXPOSURE AND CANCER INCIDENCE (CONCLUDED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Heller (1970)	Sought were abnormalities from RFEMF exposure of mitogen-stimuated cultures of human peripheral lymphocytes.	Cultures of human peripheral lymphocytes were treated for 2 days with a mitogen (PHA) to stimulate mitosis, and then exposed to pulsed 21-MHz RFEMF at a free-space-equivalent average power density of 83 mW/cm^2 for 30 minutes. Right after exposure or after 24 or 36 hours post-exposure, the cultures were fixed and scored for 6 types of chromosomal abnormalities.	Of the 600 cells scored in the unexposed control culture, 16 cells had abnormalities (of 3 of the types), for a mean of 0.016 abnormalities per cell. For the culture fixed right after RFEMF exposure, 24 of 500 cells were scored; the mean was 0.036 abnormalities per cell, a significant difference relative to the control culture. In the culture fixed 24 hours post-exposure, 131 of 2000 cells had abnormalities for a mean of 0.056 per cell, and in the culture fixed 36 hours (74 of 850 cells), the mean was 0.077 abnormalities per cell, both means significantly higher than for the control culture. The author also did similar experiments with lung cells from the Chinese hamster, and stated that the RFEMF had produced significant numbers of chromosomal abnormalities, but presented no numerical results.	Apparently only one cell culture was treated for each condition. Also, it appears that the control culture was not sham-exposed. Not stated was why different total numbers of cells were selecting for scoring. Also not clear is when the control culture was fixed following the 2-day culturing with PHA. If done immediately, then comparing the results for the two post-exposure delay periods with those for the control culture are open to question: the comparisons were made between exposed and control cultures that were not fixed at corresponding times. Little if any credence is given to the findings.
Stodolnik-Baranska (1974)	This author also sought whether exposure of mitogen (PHA)-stimulated human lymphocyte cultures to pulsed RFEMF would produce chromosomal aberrations.	After mitogen stimulation for 66 hours, cultures of human-lymphocytes were exposed to pulsed 2.95-GHz RFEMF at an average power density of 20 or 7 mW/cm^2 for durations ranging from 10 minutes to 4 hours. At 7 mW/cm^2, culture temperature stayed constant at 37°C during a 4-hour exposure; at 20 mW/cm^2, it rose by 0.5°C after 15 minutes and 1°C after 20 minutes.	At 20 mW/cm^2, the mitotic index (MI) increased monotonically with exposure duration from 12.0% at 0 minutes (no exposure) to 25.0% for 40 minutes; the rises for exposure durations of 20 and 40 minutes were significant at the 95% confidence level relative to the expected MI ranges. The results of exposure at 20 mW/cm^2 for 10 minutes, versus the number of hours of incubation, were non-monotonically higher MI values than for the non-PHA-stimulated control, but each value was within its corresponding expectation range. The numbers of chromosomal aberrations termed "dicentrics", "hyperploidy", "hypoploidy", and "breaks" for exposures of 5, 10, 15, and 20 minutes at 20 mW/cm^2 rose non-monotonically, with the largest rise for chromosomal breaks. Statistical analysis of these results was not presented.	The observation that the increases in chromosomal aberrations were not monotonic may indicate the possible presence of uncontrolled non-RFEMF factors, e.g., those related to culture-temperature variations at 7 mW/cm^2 and the observed rise in culture temperature with exposure duration at 20 mW/cm^2. Not presented was the methodology used in measuring and controlling culture temperature during RFEMF exposure. For these reasons, little if any credence can be given to the findings of this study.

TABLE 39A:CANCER INDUCTION AND PROMOTION IN MAMMALS FROM RFEMF EXPOSURE *IN VITRO*

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Chen et al. (1974)	The authors investigated whether exposure of Chinese-hamster cells to 2.45-GHz CW RFEMF would produce chromosomal aberrations. Also studied similarly were cultures of human amnion cells.	Chinese-hamster-cell cultures initially at 22°C were exposed at 50 mW/cm^2 for 10 minutes, which increased their temperature to 37°C. Other cell cultures were exposed at 85 mW/cm^2 for 4, 8, or 10 minutes, which yielded temperature increases to 37°C, 40°C, and 41°C. At exposure end, the cells were grown for 2 days, after which their growth was stopped in metaphase. The cells were then fixed and stained for chromosomal-damage analysis. In a second set, cells initially heated to 37°C were held there during exposure.	No significant difference was found between exposed and control cultures in the mean number of cells with all types of aberrations, but for some exposure conditions, the mean number of cells with specific aberrations was significantly higher than for the control cultures. However, no clear dose-dependence was evident. In the second set of experiments, cells exposed at 20 mW/cm^2 for 8 or 10 minutes, 50 mW/cm^2 for 4 minutes, or 85 mW/cm^2 for 2 or 3 minutes yielded qualitatively similar results, but again without a clear dose-dependence. One observation about the two sets was that 93% of the control cells in the first set were in mitosis versus only about 15% in the second set, and that in neither set did the various exposure conditions markedly alter the percentages. The results with human amnion cells indicated smaller chromosomal-aberration percentages than for Chinese-hamster cells, but otherwise were also qualitatively similar.	Use of a thermocouple to monitor culture temperature during RFEMF exposure may be questioned as a possible source of artifact. The statistical treatment of the data was inadequate: The authors stated the mean percentage and standard deviation (SD) for each chromosomal aberration in the sham-exposed controls, but presented the mean percentages for the RFEMF-exposed cultures without SDs, which were not calculated "in the interest of saving time". Moreover, even for the controls, the SDs were comparable to and in some cases exceeded their respective means. Because of the large variabilities in the results and the absence of dose-dependence, little if any credence can be given to the findings of this study.
Brown and Marshall (1986)	These authors sought nonthermal effects on the growth and differentiation of murine erythroleukemic (MEL) cells. They noted that in response to an inducer (HMBA), MEL cells form hemoglobin and exhibit other kinds of erythroid differentiation. Before exposure, proliferation and differentiation response to HMBA of MEL-cell cultures were characterized in 60-mm or 100-mm dishes at 37°C.	Using pairs of cellulose nitrate tubes as vessels, 12-ml cultures initially having 600,000 MEL cells in 3 mM of HMBA were exposed for 48 hours to 1.18-GHz CW RFEMF at 5.5, 11, and 22 mW/cm^2 (SARs of 18.5, 36.3, and 69.2 W/kg) at 37.4°C, with the control cultures held at the same temperature in a water bath. Four replicates were done at each RFEMF level.	The growths of exposed and control cultures were compared by measuring the elapsed times for the cells to double in number. Cell differentiation was compared by counting the percentages of cells stained by a hemoglobin-specific dye and by determining the amounts of hemoglobin produced. The results showed no significant differences in any of the three endpoints between the cultures exposed at each RFEMF level and their respective control cultures. Also, the mean values of each endpoint at the three RFEMF levels did not differ significantly from one another.	The finding of no RFEMF effect in this study is highly credible in view of the well-detailed experimental protocol and the accuracy and precision of the RFEMF dosimetry.

TABLE 39B: CANCER INDUCTION AND PROMOTION IN MAMMALS FROM RFEMF EXPOSURE *IN VITRO* (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Balcer-Kubiczek and Harrison (1985)	The presumed carcinogenic activity of RFEMF alone or in combination with benzo[a]pyrene (BP) or X-rays were examined in C3H/10T1/2 mouse-embryo fibroblasts. Also studied were the effects of the tumor promoter 12-O-tetradecacoylphorbol-13-acetate (TPA) on the transformation induction of such cells exposed to RFEMF, X-rays, or both.	Monolayers of cell cultures at 37°C were exposed to 2.45-GHz RFEMF for 24 hours at 4.4 W/kg, which raised the temperature by up to 1.2°C. Other cultures were RFEMF-exposed for 6 hours, then to X-rays at dose rate of 0.39 Gy/min for a total dose of 1.5 or 4.5 Gy, RFEMF-exposed again for 18 hours, and then treated with TPA. The controls were sham-RFEMF-exposed, treated with X-rays, or BP and either with or without subsequent incubation with TPA in acetone.	Curves of normalized cell-survival fractions (exponential scale) versus BP concentrations (linear scale) for flasks treated only with BP, or RFEMF-exposed and concurrently treated with BP, were approximately parallel and showed survival decreases with increasing BP concentration. The survival fractions for BP alone were about twice those for treatment with both BP and the RFEMF, possibly implying that the spacing between the curves was due to the RFEMF. Similar curves for cells treated with X-rays only, with or without TPA, and for cells treated with X-rays + RFEM with or without TPA, were also parallel and were spaced more that 2:1, possibly indicating that the RFEMF was the primary factor in the difference between the curves. In a graph of normalized mean transformation frequency for BP alone and for BP + RFEMF, both versus BP dose, all of the points were on the same curve, indicating that the RFEMF had no effect on transformation frequency in the surviving subpopulation of cells.	The authors concluded that the RFEMF modified cell transformation initiated by the X-rays and promoted by TPA. However, the presentation of ratios in the curves without any experimental data for the agents investigated or for the control cultures used to normalize the experimental data, renders the validity of the findings of this study questionable at best. Specifically, not presented were the numerical values of the means and SEs for the control groups in each experiment. If the control SEs were relatively large, it would indicate that uncontrolled non-RFEMF confounders existed.
Balcer-Kubiczek and Harrison (1989)	In a short communication, these authors noted that because the positive results of their previous study were observed "in a rather involved experimental protocol, our findings are difficult to interpret in terms of biohazards from microwave exposure". This later study was directed toward testing whether prolonged exposure to RFEMF could elicit and/or enhance effects of other known carcinogens.	Among the treatments were cells that were exposed to RFEMF only, exposed to X-rays only, exposed to X-rays before RFEMF exposure, and exposed to X-rays after RFEMF exposure, all TPA-treated post-exposure. The controls were cell groups: not TPA-treated, not exposed to RFEMF or X-rays, treated only with TPA in its solvent, or treated with the solvent only.	No significant differences were seen in the weighted mean plating efficiency among cells RFEMF-exposed [at 4.4 W/kg] and those sham-exposed and either subsequently cultured with TPA at 0.1 g/ml or not treated with TPA. Also, no significant differences were found in the mean survival rates for cells exposed to X-rays alone [at 1.5 Gy] or cultured with TPA after the X-ray exposure. The authors noted that these results differed from their previous findings. Bar graphs of the mean transformation frequency for the 3 TPA groups (X-rays only, X-rays + RFEMF, and RFEMF + X-rays) were much higher than for the TPA-RFEMF-only group but the error bars for those groups overlapped, possibly indicating that the differences among their mean results were non-significant.	No significant differences were seen in the weighted mean plating efficiency among cells RFEMF-exposed [at 4.4 W/kg] and those sham-exposed and either subsequently cultured with TPA at 0.1 g/ml or not treated with TPA. Also, no significant differences were found in the mean survival rates for cells exposed to X-rays alone [at 1.5 Gy] or cultured with TPA after the X-ray exposure. The authors noted that these results differed from their previous findings. Bar graphs of the mean transformation frequency for the 3 TPA groups (X-rays only, X-rays + RFEMF, and RFEMF + X-rays) were much higher than for the TPA-RFEMF-only group but the error bars for those groups overlapped, possibly indicating that the differences among their mean results were non-significant.

TABLE 39C: CANCER INDUCTION AND PROMOTION IN MAMMALS FROM RFEMF EXPOSURE *IN VITRO* (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Balcer-Kubiczek and Harrison (1991)	In another similar study, the authors investigated whether 24-hour exposure of C3H/10T-1/2 cell cultures to 2.45-GHz RFEMF would induce malignant transformation.	Such cell cultures were exposed to the RFEMF at an SAR of 0.1, 1, or 4.4 W/kg, or at 4.4 W/kg for 24 hours before or after exposure to X-rays at 0.5, 1, or 1.5 Gy. Control cultures were sham-exposed. After such exposures, cultures with or without TPA treatment were assayed for the incidence of neoplastic transformations by counting the numbers of transformed foci in culture dishes.	The neoplastic transformation incidence was low in the sham-exposed cultures, and the cultures incubated with TPA showed a slightly higher incidence than those not incubated with TPA. A plot of mean neoplastic transformation incidence (on a linear scale) versus SAR (exponential scale) for the RFEMF-exposed cultures not incubated with TPA showed no significant differences from sham-exposed cultures or any changes with increasing SAR, indicating that the RFEMF alone did not promote transformation. However, the mean neoplastic transformation incidence rose with SAR for the RFEMF-exposed cultures treated with TPA, taken as an indication that RFEMF and TPA act together to promote transformation. The mean transformation incidence versus X-ray dose for cultures not treated with TPA showed a small rise with X-ray dose, independent of either RFEMF- or sham exposure. However, the mean incidence for the TPA-treated cultures rose linearly with X-ray dose for both RFEMF- and sham-exposed cultures at about half the incidences for the latter cultures than those at the corresponding X-ray doses.	In the sham-exposed cultures, only 14 foci were found in 1494 dishes of TPA-treated cultures and 4 foci in 887 dishes of cultures not TPA-treated, a hardly robust difference. Unclear is why the numbers of dishes in the treatments varied considerably. Also, the plot of mean neoplastic transformation incidence versus SAR indicated an apparently linear rise with SAR (the points at 0.1, 1.0, 4.4 W/kg), a possibly misleading result because a linear scale was used for incidence and an exponential scale for SAR. A linear plot of those points would show a fivefold sharper rise between 0.1 and 1.0 W/kg than between 1.0 and 4.4 W/kg, a rather unusual dose-response relationship. In summary, little if any credence can be given to the findings of the three studies by Balcer-Kubischek and Harrison.
Garaj-Vrhovac et al. (1992)	Investigated were possible chromosomal aberrations in samples of lymphocytes from RFEMF exposure of whole human blood. In addition, the micronuclei in the lymphocyte samples were counted to determine the origin and relationship of micronuclei to specific aberrations produced by RFEMF exposure.	An applicator consisting of a horn antenna with an 80-cm slit was used to expose blood samples to 7.7-GHz RFEMF at 0.5 or 10 mW/cm^2 for 30 minutes, or at 30 mW/cm^2 for 10, 30, or 60 minutes, all at 22°C. Lymphocytes were then cultured for 48 hours with the mitogen PHA, and examined for chromosomal damage, which was characterized as acentric fragments, dicentric and ring chromosomes, or chromatid and chromosome breaks.	The numbers of cells that exhibited 0, 1, 2, or 3 micronuclei [fragments of the cell genome] were determined for each exposure level, and the total number of cells affected at each level was shown as a percentage of the total number of cells examined for that level. Also presented were the percentages of the cells that had each form of chromosomal aberration and the percentages of all aberrations for each exposure level. Nine of the 1000 control cells (0.9%) examined had only 1 micronucleus; the remaining 991 cells had none. For 0.5 mW/cm^2 for 30 minutes, 986 cells had no micronuclei and 14 cells had only 1 micronucleus (1.4%), a nonsignificant difference relative to the controls. The results with 10 and 30 mW/cm^2 for 10 minutes were not significantly different from the controls, but were significantly higher with 30 mW/cm^2 for 30 minutes. Seen were chromatid breaks, chromosome breaks, and acentric chromosomes, with the differences from the controls significant at 10 and 30 mW/cm^2.	Not discussed were whether control samples were sham-exposed and whether the results for the RFEMF-exposed samples from each subject were compared with control samples from the same aubject. Also absent were experimental data on how well temperature within cell preparations were controlled during RFEMF exposure, the size of the spatial temperature variations within the samples, and measurements of the incident power densities and their spatial variations. Thus, the presence of artifact cannot be ruled out. Also, the numbers of cells affected were small relative to the totals examined. Therefore, little if any credence is given to the findings of this study.

TABLE 39D: CANCER INDUCTION AND PROMOTION IN MAMMALS FROM RFEMF EXPOSURE *IN VITRO* (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Ivaschuk et al. (1997)	Investigated was whether PC12 (pheochromocytoma) rat cells treated with nerve growth factor (NGF) and exposed to time-domain-multiple-access (TDMA)-modulated 836.55 MHz-RFEMF would affect the transcription of immediate-response genes *c-jun* and *c-fos*.	Monolayer cell cultures seeded onto Petri dishes and treated with NGF were exposed with a TDMA transmitter that fed 2 TEM cells, with four dishes on the septum of each cell. The duty cycle of the pulsed carrier was 33%. The time-averaged spatial mean SAR was 0.97 W/g per mW/cm^2. Exposures were at 0.09, 0.9, and 9 mW/cm^2 for durations of 20, 40, or 60 minutes with 20-minute on/off intervals for the latter two durations.	RNA was isolated, size-fractionated on gels, transferred to nylon membranes, cross-linked with ultraviolet in buffer containing cDNA probes for *c-fos* and *c-jun*, and labeled with 32P. Use of a radioanalytic imager on the "Northern blots" (with GPDH as the internal standard) yielded mRNAs of 2.2 kb for *c-fos*, 2.7 and 3.2 kb for *c-jun*, and 1.4 kb for GPDH. The results were expressed as ratios of the dpm for *c-fos* or *c-jun* to the dpm for GDPH for each RFEMF-exposed or sham-exposed sample, and then each as the ratio of its results (E/C). For *c-fos*, the mean E/C values for 0.09 and 0.9 mW/cm^2 were essentially the same: 1.03 ± 0.15 and 1.03 ± 0.11; for 9 mW/cm^2, the E/Cs ranged from 0.58 to 1.01, with a mean of 0.86 ± 0.17, a nonsignificant decrease in transcript level. The results for *c-jun* were similar except for 20 minutes of exposure at 9 mW/cm^2, which yielded a significant decrease in transcript level (39%).	The frequency and modulation of the TDMA transmitter conformed with the North American digital cellular telephone standard. When the carrier is on, it consists of digital data of constant amplitude, with the phase of each bit shifted by ± 45° or ± 135° relative to the preceding bit as determined by the data. The authors noted that Phillips also found no effect on *c-fos* expression in NGF-treated PC12 cells exposed to 60-Hz magnetic fields up to 100 T (unpublished data), in contrast with Phillips et al. (1992) in which large *c-fos* increases were seen in CCRF-CEM T-lymphoblastoid cells exposed at 100 T.
Stagg et al. (1997) [Companion study to Ivaschuk et al. (1997)]	Investigated was whether exposure of rat glioma cells to the TDMA-modulated 836.55-MHz RFEMF would promote tumor growth by altering DNA synthesis and elevating cell proliferation. Normal glial cells were also RFEMF-exposed.	C6 glioma cells in log-phase were incubated for 24 hours, tagged with tritiated thymidine (TdR), dissociated, adjusted to 80,000 cells per ml, and transferred in 0.5-ml volumes to 48-well plates or 60-mm culture dishes. Exposures at each RFEMF level were begun 18-24 hours following transfer. At 0.9 mW/cm^2 the SAR was 5.9 W/g for 48-well plates and 1.5 W/g for 60-mm dishes. Normal glial cells from 17-day-old rat fetuses were similarly treated.	The TdR results were tabulated as the mean ratios (E/S) ± SE of the counts after 24-hour exposure at 0.59, 5.9, or 59 W/g to the counts for sham exposure, and E/S ± SE after 4 hours of exposure at 5.9 W/g. The only significant result (by t-test) was for exposure at 5.9 W/g for 24 hours (P=0.026). That finding, presented in more detail, showed the results for eight independent assays of E/S at that RFEMF level with an error bar for each mean. Those mean E/S values ranged from about 80% (20% below the 100% line) to about 140% (40% above that line), with 3 of the 4 E/S values above the line tagged as significant. Also displayed was the mean E/S and error bar for the combined data set of RFEMF exposures, and similarly for the combined sham exposure set. The mean for RFEMF group was about 110%, the mean for the sham was 100%, and their error bars were non-overlappng, yielding the stated P = 0.026. The analogous results for normal glial cells exposed for 24 hours at 5.9 or 59 W/g, showed no significant differences in mean E/S.	The findings were negative, but some aspects of this study are unclear. First, the cell cultures were plated onto either 48-well plates or 60-mm culture dishes, but the authors did not discuss why the numbers of assays and their total sample sizes were fewer than the numbers in the 48-well plates or the 60-mm culture dishes, and differed for the three RFEMF levels used. Were the results for some of the wells or those in culture dishes discarded, and if so, why? Second, why were the mean E/S values for the sham exposures all 1.00, but with SEs that differed from one another? These points, and particularly the absence of the E-data and S-data, from which the E/S values and their SEs were derived, would tend to diminish the degree of validity of the negative findings.

TABLE 39E: CANCER INDUCTION AND PROMOTION IN MAMMALS FROM RFEMF EXPOSURE *IN VITRO* (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Cain et al. (1997) [Companion study to Ivaschuk et al. (1997]	Studied was whetherTDMA-modulated 836.55-MHz RFEMF and TPA are copromoters that can enhance the growth and focus formation of murine C3H/10T1/2 fibroblasts in coculture with parental C3H/10T1/2 cells. It was noted that parental cells suppress growth of the transformed phenotype of daughter 10e cells by preventing 10e cells from growing in multilayered foci, and that TPA relieves that suppression by the 10T1/2 cells and thereby promotes focus formation.	Cultures of 10T1/2 and 10e cells were grown respectively in T-75 and T-25 flasks at 37±0.2°C. After 3-4 days, 70%-80% confluent 10T1/2 and 10e cells were seeded into 60-mm petri dishes as cocultures of 1600 10T1/2 cells and 400 10e cells and as the same cell-percentage cocultures plus TPA. Cocultures with and without TPA were exposed for alternating 20-minute on/off periods until day 29. Treatment with TPA was done on days 2, 9, 16, and 23.	Sham-exposed cells stained and assayed for focus formation yielded mean numbers of foci per dish of 10.1 ± 1.2, 28.3 ± 2.8, and 32.8 ± 2.8 for TPA doses of 10, 30, and 50 ng/ml. The values for the cultures RFEMF-exposed at 1.5 W/g were 9.8 ± 1.1, 27.3 ± 3.2, and 35.6 ± 3.5. Thus, a linear increase with TPA dose.was evident for both, but no significant differences were found between the sham-exposed and RFEMF-exposed cultures at corresponding TPA doses. Similar results were obtained for mean foci area and mean foci density, again with no significant differences between the RFEMF-exposed and sham-exposed cultures. At 15 W/g, the mean numbers of foci per dish were smaller and increased nonlinearly with TPA dose, but with no significant differences between RFEMF-exposed and sham-exposed cultures. Similar results were found for the other endpoints.	As did Ivaschuk et al., Cain et al. remarked that their negative findings are in contrast with their earlier positive results [Cain et al. (1993)] for the same endpoints, using a 60-Hz magnetic field. The overall conclusion from this and the two companion studies is that exposures in vitro of the specific cell types to the TDMA-modulated 836.55-MHz RFEMF neither initiates nor promotes or co-promotes (with TPA) tumor growth. A few questions are raised about some aspects, such as those for Stagg et al. (1997), but considering the studies collectively, the validity of this conclusion is high.
Malyapa et al. (1997a)	The authors sought to determine whether exposure to 2.45-GHz RFEMF of cultured mammalian cells would cause DNA damage in human glioblastoma U87MG cells or in mouse C3H 10T1/2 cells. The "alkaline comet assay" was used to determine DNA-fragment migration under electrophoresis in terms of "comet length" [CL] and "normalized comet moment" [NCM].	The exposure facility was a set of parallel-plate, radial-transmission-line systems (RTLs) each fed with a central conical antenna. The RTL set was capable of concurrently exposing large numbers of culture flasks. Groups of T-75 flasks having monolayers of each cell type in exponential growth phase were exposed to 2.45-GHz RFEMF at 0.7 W/kg for 2, 4, or 24 hours and cells were assayed for CL and NCM. As a positive control, cell preparations were exposed to gamma radiation and assayed.	The growth rates of each cell type after RFEMF exposure were found to be essentially the same as for their sham-exposed controls. Plots of NCM and CL for U87MG cells after RFEMF exposure showed no significant differences in either endpoint relative to sham-exposed cultures or with increasing exposure duration. A similar lack of significant differences was found for C3H 10T1/2 cells. In the gamma-irradiation positive-control experiments, the frequency distributions of NCM and CL intensity were shown as bar graphs of counts per minute at each NCM and CL value and at gamma-ray doses of 0, 0.3, 0.6, 1, 3, and 5 cGy. The set of NCM bars at each successively higher dose showed a monotonic increase with dose. The results for the CL were similar, but not as pronounced. There was no evidence of DNA migration in non-irradiated cells of either type.	The authors, in their discussion, noted that their gamma-ray dose responses were consistent with the dose-response relationships found by others. They also noted that their negative findings differed from the positive findings of Lai and Singh (1995, 1996), who had suggested that the findings could be an indirect effect requiring some post-exposure time to develop. However, Malyapa et al., citing Williams (1996), stated that this concept is controversial and does not conform to the conventional concepts of repair of DNA damage from other forms of radiation. They also remarked that the Olive et al. (1992) comet assay method used in their experiments was as sensitive as the method used by Lai and Singh.

TABLE 39F: CANCER INDUCTION AND PROMOTION IN MAMMALS FROM RFEMF EXPOSURE *IN VITRO* (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Malyapa et al. (1997b) [Companion study to Malyapa et al. (1997a)]	The authors did a similar study with glioblastoma U87MG and C3H 10T1/2 cells, but with exposure to FMCW RFEMF centered on carrier frequency 835.62 MHz and CDMA-modulated RFEMF at carrier frequency 847.74-MHz. Both cell types were treated in exponential growth phase, but C3H 10T1/2 cells in the plateau phase were also studied. As before, cultures were irradiated as well with gamma rays as a positive control.	Multiple RTLs were used for sham- and RFEMF exposures for 2, 4, or 24 hours at an estimated mean SAR of 0.6 ± 0.3 W/kg for both FMCW-modulated and CDMA-modulated RFEMF. After exposure, the cultures were analyzed for DNA damage with the alkaline comet assay of Olive et al. (1992), using NCM and CL as endpoints.	The results with U87MG cells in exponential-growth phase indicated no significant differences in NCMs or CLs for exposure to the modulated RFEMF of either type versus exposure duration. The bar-graph frequency distributions showed no significant increases relative to sham exposure. The results for C3H 10T1/2 cells in exponential growth were similarly negative, but as before, those for the gamma-ray exposures exhibited monotonic increases in both CLs and NCMs with dose. Similarly treated C3H 10T1/2 in the plateau phase of growth also showed no significant differences in CLs and NCMs. In addition, C3H 10T1/2 cells in both growth phases were RFEMF-exposed or sham-exposed for 2 hours, but then incubated for 4 hours at 37°C before processing for the comet assay. [See Notes & Comments.] Once more, no significant differences in NCMs or CLs were seen relative to sham exposure of the cells in either phase. As another positive control, separate experiments with C3H 10T1/2 cells labeled with *BrdU and exposed to fluorescent light yielded a significant increase in NCM and CL.	*BrdU (5-bromo-2'-deoxyuridine) is known to sensitize proliferating cells toward DNA damage from X-rays or ultraviolet light. The experiment involving 4-hour post-exposure incubation before processing cells for the comet assay was performed to test (under in vitro conditions) the controversial indirect-effect concept of Lai and Singh mentioned under "Notes and Comments" of the companion paper. The absence in that experiment of significant differences does not appear to support that concept. As noted earlier about the two Lai and Singh studies, it seems likely that significant brain autolysis may have occurred during the period between mice euthanization after exposure end and the start of cell-culture preparation for assay. Thus, it is unclear whether such studies involving RFEMF exposure in vivo could be done without introducing non-RFEMF-related factors.

TABLE 39G: CANCER INDUCTION AND PROMOTION IN MAMMALS FROM RFEMF EXPOSURE *IN VITRO* (CONCLUDED)

5 MUTAGENESIS AND GENOTOXICITY IN MICROORGANISMS AND FRUIT FLIES

5.1 MICROORGANISMS

Blackman et al. (1976) investigated whether exposure of cultures of the WWU strain of bacterium Escherichia coli (E. coli) to RFEMF would induce mutations therein. This strain was selected because it requires arginine (and other constituents) in the nutrient medium for growth. WWU-strain cells that can grow in the absence of any of the constituents are regarded as mutants.

Cultures of E. coli were grown for 90 minutes at 37°C in either of two media, one on Nutrient Broth plates containing arginine (and other necessary constituents) and the other in A-1 Media [used for determining mutation caused by decay of tritium incorporated in E. coli], but lacking arginine. The concentration was adjusted to 50 million cells per ml, and 1.5-ml samples were placed in tissue culture dishes. Such dishes were exposed at 35°C to 1.70-GHz or 2.45-GHz CW RFEMF in a temperature-controlled anechoic chamber for a duration (mostly 3.5 hours) long enough to include at least one full DNA replication cycle to ensure that each part of the genome was replicated. The exposures to 1.7-GHz RFEMF were done in the near field, at 88 V/m. The equivalent free-space power density was 2.05 mW/cm^2 and the SAR, estimated from probe measurements, was 3 W/kg. The exposures to 2.45-GHz RFEMF were in the far field at 10 and 50 mW/cm^2. By calorimetry, the SARs were 15 and 70 W/kg. The control cultures were placed on the stand with the cultures to be exposed, but were shielded from the RFEMF with aluminum foil. Before and after treatment, counts were done to assess the number of colony-forming units/ml and the mutation frequency at the arginine locus.

As a positive control, cell samples were exposed to ultraviolet light, and plots of the survival percentages and the numbers of mutants/ml versus UV dose showed the expected exponential survival curve and increase in arginine-independent mutants.

The growth in the total cell populations in the arginine-containing and arginine-lacking media during RFEMF- and sham exposure were expressed as mean numbers of cell doublings relative to the initial populations, and were compared statistically. Also compared were the concentrations of arginine-independent mutants in these populations. The difference in each endpoint between cultures exposed to 1.7-GHz RFEMF at 88 V/m (3 W/kg) and sham-exposed cultures was nonsignificant. This was also true for cultures exposed to 2.45-GHz RFEMF at 10 mW/cm^2 (15 W/kg), usually for 3.5 hours, or at 50 mW/cm^2 (70 W/kg) for 3.2 hours. However, cultures exposed to 2.45-GHz RFEMF at 50 mW/cm^2 (70 W/kg) for 4.0 hours instead of 3.5 hours yielded a significant increase in the cell doublings and a significant decrease in the concentrations of mutant cells. The authors, citing a previous study (Blackman et al., 1975)], regarded the latter results solely as a thermal effect. They concluded that the RFEMF exposures produced no mutagenic activity.

Dutta et al. (1979) sought possible mutagenic effects of RFEMF in strains TA-1535, TA-100, and TA-98 of Salmonella *typhimurium* bacteria. All three are prokaryotic strains (not having true nuclei); TA-1535 and TA-100 are used frequently to test for base-pair substitutions, and TA-98 for frame-shift alterations. The authors also sought mutagenic effects in diploid strain D_4 of Saccharomyces *cerevisiae*, a primitive eukaryotic yeast (having a true nucleus) of intermediate genome complexity between prokaryotic and mammalian eukaryotic cells, a strain frequently used to test for genetic conversion and mitotic recombination.

Exposures to 2.45-GHz CW RFEMF (S-band) and to several frequencies of pulsed RFEMF (1-μs pulses at 1000 pps, 0.001 duty cycle) in the range 8.5-9.6 GHz (X-band) were performed in separate anechoic chambers at controlled temperature and humidity. Samples of S. *cerevisiae* in log phase on petri plates were exposed for 2 hours at 30°C to the S-band CW RFEMF at 20 mW/cm^2 or at 29°C to the pulsed X-band RFEMF in the range 8.5-9.6 GHz at average power densities up to 45 mW/cm^2. Samples of S. *typhimurium* in log phase were exposed to the same levels of CW or pulsed RFEMF, but for 90 minutes at 37°C or 35°C, respectively. The corresponding SARs were both about 40 W/kg.

Following post-exposure incubation for 72 hours, yeast cells were scored for numbers of cells converted to adenine- and tryptophan independence per 100,000 surviving cells. Similarly, after post-exposure incubation for 48 hours, bacterial cells were scored for the total number of colony-forming units per milliliter and the number of revertant cells per 10^8 survivors. The Genetic Activity Index (GAI) was calculated for both cell types, with 1.0 or below signifying no effects.

The results for the yeast cells exposed to the S-band RFEMF showed little change in genetic events relative to controls. For the X-band exposures, their survival percentages were higher at 5 mW/cm^2 than at 1 or 45 mW/cm^2, indicating that uncontrolled non-RFEMF factors may have been present. The bacterial cells exposed to the S-band RFEMF also showed little change in genetic events. For strain TA-100 exposed to X-band, the GAIs for reversion were all below 2.0. At 10 mW/cm^2, TA-1535 and TA-98 generally had GAIs well below 2.0 except for TA-1535 with 9.0 GHz: a GAI of 2.57. However, those cells had a 100% survival rate.

The authors stated that positive-control experiments were done with the chemical mutagen ethyl methanesulfonate (EMS). They gave no details, but indicated that the results thereof and historical control data in the literature were the basis for using the genetic activity index (GAI), defined as the ratio of the frequency of genetic events in the treated population to that in the control population.

The results for exposure of the yeast to 9.0-GHz pulsed RFEMF at 9 levels in the range 1-45 mW/cm^2 used as average power densities (peak power densities of 1-45 W/cm^2) were tabulated. They indicated that the numbers of conversions to adenine independence varied nonmonotonically with increasing power density. Moreover, the numbers of conversions for the corresponding sham-exposed samples also increased nonmonotonically. At 1 mW/cm^2 for example, there were 5.6 gene convertants per 100,000 survivors versus 5.7 for the corresponding sham-exposed samples; at 30 mW/cm^2, the respective numbers were 2.2 and 3.6; and at 45 mW/cm^2 they were 48.0 and 41.0. Clearly, those increases were due to factors other than RFEMF exposure. The results for conversion to tryptophan independence were qualitatively similar.

Percentage survival rates and GAIs for exposure of the yeast versus RFEMF frequency at 1, 5, and 45 mW/cm^2 were also tabulated. Again the variations were nonmonotonic in both RFEMF frequency and level, but the survival rates at 45 mW/cm^2 were all lower than for 1 and 5 mW/cm^2, results ascribed by the authors to large rises in sample temperature (at least 12°C above ambient). The GAIs were all less than 2.0 except for 9.4 GHz at 45 mW/cm^2, for which the GAI was 2.17 for conversion to adenine independence but only 0.97 for conversion to tryptophan independence.

The results for exposure of the TA-100 base-pair-substitution mutant strain of S. *typhimurium* to 8.5-9.6 GHz pulsed RFEMF at 10 and 45 mW/cm^2 were tabulated. At 10 mW/cm^2, the number of reversions varied nonmonotonically with frequency, ranging from a low of 27 per 100 million cells at 8.8 GHz to a high of 35 per 100 million cells at 9.4 GHz. However, number of reversions for the sham-exposed cells also varied nonmonotonically with frequency, with a low of 17 per 100 million cells also at 8.8 GHz to a high of 44 per 100 million cells at 9.6 GHz (not 9.4 GHz). The corresponding GAIs ranged from 0.61 at 9.6 GHz to 1.38 at 8.8 GHz. At this RFEMF level, the survival rates ranged from 88% to 100%. Similar reversion results were shown for exposure at 45 mW/cm^2, with GAIs ranging from 0.43 at 9.0 GHz to 1.91 at 8.8 GHz, but the percentage survival rates were all lower than at 10 mW/cm^2. The results for the TA-1535 base-pair-substitution mutant strain at 10 mW/cm^2 were qualitatively similar to those for the TA-100 strain at that level. The GAIs ranged from 0.87 at 9.6 GHz to 2.57 at 9.0 GHz.

The GAI-versus-frequency results for the TA-98 frame-shift mutant strain at 10 mW/cm^2 ranged from 0.42 at 9.4 GHz to 1.91 at 9.0 GHz. (No results for either the TA-98 or TA-1535 strain exposed at 45 mW/cm^2 were presented.)

Also, based on previous observations, the authors noted that: values of the GAI that do not exceed 1.0 signify no RFEMF-induced mutagenesis, values between 1.0 and 2.0 are within the normal fluctuation range, values between 2.0 and 3.0 are "suspect", and values exceeding 3.0 definitely indicate mutagenesis.

The authors, noting that all of the GAIs were less than 3.0, concluded that the exposures of the yeast Saccharomyces *cerevisiae* or the bacteria Salmonella *typhimurium* to 2.45-GHz CW RFEMF or to pulsed RFEMF in the X-band range (8.5-9.6 GHz) at average power densities of 30 mW/cm^2 or higher reduced their viability but did not reliably induce genetic changes,. However, they did not present any statistical treatment of the results, such as numbers of samples treated at each frequency and RFEMF level, and the variances in the data, particularly the large variations in the results for the various sham-exposed samples. Thus, although the findings of this study were negative, non-RFEMF factors may have been present.

Dardalhon et al. (1979) used two haploid strains and a diploid strain of S. *cerevisiae* to determine the effects of temperature and RFEMF on survival and on the induction of mitotic recombination or cytoplasmic "petite" mutations. These two haploid strains were genetically deficient in synthesizing certain amino acids and were of opposite mating types that form stable heterozygotic diploid cells; they were used to test for effects of RFEMF on zygote formation. The diploid strain has two alleles that can be distinguished by their color; treatment by a mutagen yields a color change, thus permitting detection of genetic effects, including mitotic recombination induction.

For exposure to RFEMF, cells in saline suspension were collected with a millipore-filter disc, which was placed on the surface of agar within an open petri dish. Each dish was mounted on a foamed-polystyrene block, and the exposures were done from above at 2 or 10 mm from the face of a horn terminating a waveguide. Both sites were within the near field of the horn. The exposures were at several power densities up to 60 mW/cm^2 for 180 or 330 minutes at 20°C to 70.5-GHz or to 73-GHz CW RFEMF. Control cultures were sham-exposed, and other cultures were placed in a chamber held at 30°C, 37°C, 42°C, 47°C, or 52°C for 330 minutes.

To estimate the RFEMF-induced temperature increases, water evaporation from solidified agar was measured for exposures (at the 10-mm site) to 70.5-GHz RFEMF at 60 mW/cm^2 and a baseline temperature of 20°C for several durations up to 180 minutes. The results were compared with those from conventional heating at 20°C, 30°C, and 37°C for the same durations. For the latter treatments, the graphs of agar mass versus time were linear, with slopes that were successively more negative (higher water-loss rates) with increasing temperature. The corresponding graph for RFEMF exposure was also linear, with a negative slope between those for heating at 30°C and 20°C but closer to the slope for the latter. From the results, the authors concluded that RFEMF exposure at a baseline temperature of 20°C increased sample temperature by no more than 2-3°C. (However, specimen-temperature increases from exposure at the 2-mm site were most likely larger.)

The ratio of cell survival of treated samples to that of controls was plotted versus duration for exposure of the diploid strain at the 10-mm site to 70.5-GHz or 73-GHz RFEMF at 15 or 60 mW/cm^2 for up to 180 minutes. For all exposure conditions, the relative survival curves exhibited ratios that changed little from unity with time.

With the color-change technique noted above, the percentages of altered colonies were found to be "practically nil" after exposures under the four conditions above, indicating that the RFEMF had no effect on nuclear DNA. The results were also negative for induction of cytoplasmic "petite" mutations, an indication that the RFEMF had no adverse effects on mitochondrial DNA. (The results for exposures at the 2-mm site were not presented.) Conventional heating at 30°C, 42°C, and 47°C for the same durations also had no effect on the relative survival ratios, percentages of altered colonies, or "petite" mutations. However, marked decreases in the percentages of survival and increases in the percentages of altered colonies and "petite" mutations were obtained at 52°C.

The two genetically-deficient haploid strains were also tested for zygote formation by mixing suspensions containing 100 million cells per ml of each strain in equal volumes, depositing 0.05-ml samples of the mixture on millipore filters, and setting each filter on a solid complete-growth medium in an open petri dish. Each dish was exposed at the 2-mm or 10-mm site to 70.5-GHz RFEMF at 6, 15, or 60 mW/cm^2 for 330 minutes. After treatment, the samples were grown in a medium containing no amino

acids, thus allowing only the growth of zygotes and heterozygotic diploid cells in which the genetic deficiencies of one strain were complemented by the normal alleles of the other strain.

The results at each power density for both exposure sites were graphed as ratios of zygote formation for the sample treated to zygote formation for the control sample. For the 2-mm site, the ratios were 1, 1.75, and 3.1, respectively for 6, 15, and 60 mW/cm^2. For the 10-mm site, the corresponding ratios were about 1.2, 1.2, and 1.4. The ratios were much larger for samples conventionally heated for 330 minutes at 20°C, 30°C, and 37°C, about 1, 20, and 57, respectively. The authors surmised from these results and from the increases in culture temperature discussed above that RFEMF exposure at 60 mW/cm^2 would be equivalent to heat treatment that raises culture temperature by no more than 3°C. They also surmised that culture temperature would increase by less than 0.5°C from exposure at 10 mW/cm^2.

The authors concluded that exposure to millimeter waves under the stated conditions does not induce lesions or genetic effects in cellular DNA of S. *cerevisiae*, whereas intense conventional heating does. As remarked by them, the findings of Webb and Dodds (1968) on inhibition of bacterial growth by exposure to 136-GHz RFEMF cannot be explained by direct action of RFEMF on cellular DNA.

Dardalhon et al. (1981) studied various ultraviolet-sensitive strains of procaryote E. coli and eucaryote S. *cerevisiae*, but at 9.4 GHz (a frequency used in microwave thermography and other applications), and at 17 GHz (where maximal effects on the relaxation of water molecules at room temperature can be expected). The corresponding wavelengths were 3.19 cm and 1.76 cm. In addition, sought further were possible effects in the range 70-75 GHz. For the latter exposures, the samples were placed at the 2-mm site used in the previous study. A horn was used for the 9.4-GHz exposures, but the exposure sites were at 5 mm and 3 cm from the horn; these two sites were also used for 17 GHz, but the antenna was the open end of a waveguide instead of a horn.

The authors determined SARs in samples at the exposure sites by using the observation that zygote formation in S. *cerevisiae* is very sensitive to changes in temperature. For 9.4 GHz, the normalized SAR at the 5-mm site was 0.38 W/kg per mW/cm^2. For 17 GHz, it was 0.56 W/kg per mW/cm^2. (The values at the 3-cm site were not given for either frequency.) For 70-75 GHz, the normalized SAR at the 2-mm site was 0.15 W/kg per mW/cm^2. Most exposures were for 30 minutes. Control samples were sham-exposed. For positive controls, samples were exposed to 254-nm ultraviolet radiation.

Six strains of E. coli were studied, four of which were deficient in DNA repair. The fifth was a wild-type DNA-repair-proficient strain and the sixth was a mutant strain that requires tryptophan for growth. Most of the fractional survival rates for the DNA-repair-deficient strains and the DNA-repair-proficient wild strain after exposure were between 0.90 and 1.0. A few were larger than 1.0 (maximum 1.1 for 9.4 GHz at 23 W/kg), and a few others were less than 0.90 (minimum 0.80 for 74 GHz at 9 W/kg). The authors therefore concluded that the RFEMF had no appreciable effects on survival, even in the repair-deficient mutants. They also noted that survival fractions of the wild-type and mutant cells decreased strikingly after conventional heating for 30 or 60 minutes at temperatures above 50°C, with the mutants more sensitive than the wild-type cells.

For tryptophan-dependent E. coli, the ratios of the number of mutations to tryptophan independence induced by 30-minute exposures to RFEMF in the range 70-75 GHz (9 W/kg) to the number of spontaneous mutations was plotted versus RFEMF frequency. These ratios varied non-monotonically between 0.5 and 1.0. For exposures to 17 GHz at the 3-cm site (no SAR given), the mutation ratio varied with exposure duration: from about 1 for 30-minute exposures, to a minimum of 0.2 for 1-hour exposures, to 1 for 2-hour exposures, and to 1.5 for 20 hours of exposure. The authors noted that the standard error was ± 1.25, so the values were not significantly different from the spontaneous background.

The S. *cerevisiae* studied were a haploid wild-type strain and a DNA-repair-deficient mutant strain for DNA effects, a diploid strain for the detection of genetic alterations, and the latter and another diploid strain for sporulation effects. After sham exposure, the survival fraction of the wild-type strain was 0.81. Exposure (30 minutes) to 9.4 GHz (23 W/kg) yielded a survival fraction of 0.96. For exposures to 17 GHz at the 5-mm site (28 W/kg) and the 3-cm site (SAR not given), the survival fractions were 1.15 and 0.89,

respectively. For exposures to millimeter waves (9 W/kg), the survival fraction varied non-monotonically with frequency from 0.80 at 75 GHz to 1.16 for 70 GHz. The differences among values were stated to be nonsignificant. The percentages of cytoplasmic petite mutations in the haploid strain after RFEMF exposure did not differ significantly from the percentage of spontaneous mutations.

The results for sham exposure on the histidine-dependent strain of S. *cerevisiae* for exposure durations of 30, 60, and 120 minutes, respectively were 2.6, 1.4, and 1.4 reversions to histidine independence per 100 million survivors. The values for RFEMF exposure ranged from 1.0 for 60 minutes of 9.4 GHz to 6.1 for 30 minutes of 73 GHz, regarded by the authors as in the range of normal fluctuation.

Exposure of the diploid strain to 17-GHz RFEMF at 50 mW/cm^2 at the 5-mm site (28 W/kg) for up to 24 hours did not yield significant effects on the relative survival fraction or induction of cytoplasmic petite mutations or of genetically altered colonies (including mitotic crossovers). By contrast, conventional heat treatments at 47°C for 24 hours or at 52°C for 2 hours markedly decreased the relative survival and increased the numbers of petite mutations and altered colonies.

To determine the effects of RFEMF exposure on sporulation, samples of one of the two diploid strains were sham-exposed or exposed for 48 hours during sporulation to 9.4 GHz (23 W/kg) or to 17 GHz (28 W/kg). The sporulation efficiencies for the three treatments did not differ significantly. The other diploid strain was similarly treated but for 72 hours during meiosis. Again, the differences among the treatments were not significant.

As for the previous study, the lack of statistical treatment of the results diminishes confidence in the findings.

Anderstam et al. (1983) also investigated whether RFEMF is mutagenic for E. coli and S. *typhimurium* (using a total of 11 strains). Exposures were to separate near-field 27.12-MHz CW electric and magnetic fields, the former between parallel capacitor plates and the latter within a Helmholtz coil. Other exposures were far-field 2.45-GHz RFEMF amplitude-modulated at 100 Hz (using a magnetron source), and 3.07-GHz pulsed RFEMF (2-μs pulses at 500 pps, 0.001 duty cycle).

For adequate biological sensitivity, most sample volumes were 10 ml each. Since most media contain substances that can be autooxidized in the presence of metal ions, possibly forming toxic components, sample containers were made of polytetrafluoroethylene (PTFE) because tests showed that the metal-ion content of PTFE is very low and because such containers can be sterilized readily in an autoclave.

For electric-field exposure to 27.12 MHz, each PTFE container was put inside a larger container made of polymethylmethacrylate (PMMA) through which flowed thermostatically controlled water. By this means, sample temperature during exposure was held to within ± 0.3°C of that of an unexposed sample. The larger container was inserted between 20-cm-square vertical capacitor plates spaced 4 cm apart, with a 2.5-mm air gap on each side of the container. With this geometry, the electric field was calculated and the SAR of the sample was determined from the measured dielectric properties of the sample at that frequency. With 400 V (rms) applied to the plates, the field (in air) was 10 kV/m, the field within a sample was 72 V/m, and the SARs in S. *typhimurium* and E. coli samples were 3 W/kg and 2.5 W/kg, respectively.

The 27.12-MHz magnetic field was produced with Helmholtz coils 22 cm in diameter and spaced 11 cm apart. The exposures were done for each cell sample within a glass tube (6.5 cm long and inner radius 7 mm) and the tube axis parallel to the magnetic field, or with 10 ml of each sample within a groove cut in a PTFE disk at radius 7.2 cm and disk-plane perpendicular to the magnetic field. SARs for both species of bacterium at 20 A/m for the disk and tubular containers were respectively 20 W/kg and less than 0.15 W/kg.

The rectangular PTFE container above was also used for exposures to the amplitude-modulated 2.45-GHz RFEMF and the pulsed 3.07-GHz RFEMF, but the container was placed 40 cm from a horn (far field). Thermocouple measurements of temperature were made at 15 locations on the container

immediately before and after exposures to 2.45 GHz at 900 mW/cm^2 for 30 seconds or 3.1-GHz RFEMF at 290 mW/cm^2 (average) for 60 seconds. Cooling curves were used to determine the corresponding SAR at each location, from which the local value of electric field was calculated and normalized to the mean for all locations. The normalized electric-field values ranged from 0.70 to 1.26 for 2.45 GHz and 0.88 to 1.08 for 3.07 GHz. Thus, the variation of power density over the sample area was less than ± 3 dB and the mean SAR for 200 mW/cm^2 was about 100 W/kg (0.5 W/kg per mW/cm^2) for either frequency.

Used for the study of forward mutations were three strains of S. *typhimurium* sensitive to arabinose, and two strains of E. coli, one dependent on streptomycin and the other sensitive to rifampicin. Back mutations were studied in two histidine-dependent S. *typhimurium* strains, two tryptophan-dependent strains of E. coli, and one strain of E. coli dependent on tyrosine. Prophage induction was investigated in two strains of E. coli, using a streptomycin-resistant strain as an indicator. Exposures to each type of RFEMF at the levels above were for various durations in the range 1-7 hours. In most experiments, air was bubbled through the cell suspensions during exposure for stirring. Also investigated were the combined effects of the RFEMF with ultraviolet light (UV) at a dose rate of 0.033 W/m^2 given before or after RFEMF exposure.

The various treatments of each strain of the two bacteria species (11 strains of bacteria, four types of RFEMF treatment, and combined UV and RFEMF treatment) were tabulated in the paper, as were the results of statistical analyses of the data. For some RFEMF treatments, some strains exhibited higher growth and others lower growth than their respective controls. Many of these changes were nonsignificant, but the overall trend was toward RFEMF-induced increase in growth. The authors could not ascribe the growth changes solely to temperature increases, stating that: "...changes of temperature in unexposed samples by 0.5°C certainly provoked changes of the growth rate, but these changes were smaller than observed after microwave exposure." However, another finding was that the differences in mutant counts (both increases and decreases) induced by RFEMF were mostly nonsignificant.

In this paper, the authors presented considerable quantities of data, statistical treatments thereof, and much discussion of their interpretations based on a variety of considerations difficult to assess, mostly because the results were mixed, e.g., the higher and lower growth in different strains noted above. Among their overall conclusions based on their data was that cancer risk and risk of heritable damage from occupational microwave exposure in Sweden is less than one case per year. However, even though they studied the effects of combining RFEMF exposure with UV, not clear is whether they had used UV or any other known mutagenic agent alone as a positive control, to assess the adequacy of their methodology, and specifically whether significant non-RFEMF factors were present.

5.2 FRUIT FLIES

Pay et al. (1972) exposed three groups of five Oregon-R, wild-type male Drosophila *melanogaster* for 45 minutes to 2.45-GHz RFEMF, each group within an acrylic capsule placed in the near field of a horn within an anechoic chamber. One group each was exposed at 2.1, 2.75, and 3.0 kW forward power. The corresponding mean power densities, calculated from measurements at 200 cm (far field), did not exceed 4.6, 5.9, and 6.5 W/cm^2. The exposures were done within 24 hours after eclosion. For each RFEMF level, a capsule of flies was sham-exposed as controls. To exercise control over generation times, two growth temperatures were used prior to exposure: The flies for the 2.75-kW and 3.0-kW groups were grown at 22.5°C and those for the 2.1-kW group at 21.0°C. The later generations derived from these exposure groups were grown at the same respective temperatures.

Within 30 minutes after exposure, each male was placed in a vial with two virgin females of the Muller-5 type. After this initial mating, each male was placed in a new vial together with two new virgin Muller-5 females every 24 hours for 15 days after exposure, thereby obtaining 15 "Standard-Muller-5-Cross (Basc)" broods from each wild-type male fly. On day 8 after each mating, the Muller-5 females were removed and the number of days to emergence of the first adult flies in each brood was recorded as the generation time. All F1 (first-generation) broods were counted and sexed on day 17 of growth (before second-generation eclosion); if no adult F1 flies were present on that day, the growth stage was noted.

Forty-five F1 male-female pairs from exposed males and 15 male-female pairs from sham-exposed males were selected at random each day from the 2.75-kW and 3.0-kW groups, and each pair was placed in a new vial for mating to produce an F2 generation. Those F2 generations were examined on day 17 for wild-type males because their presence would rule out the possibility of sex-linked lethal recessive mutagenesis on the X-chromosome of the parent (P1) males exposed to RFEMF. Also, such a recessive mutation would cause lethality in F2 hemizygous (having only one of a pair of genes for a specific trait) wild-type males.

All of the P1 males in the control, 2.1-kW, and 3.0-kW groups survived for use in the brood studies, but 2 of the 5 P1 males of the 2.75-kW group died. The F1 flies from the 2.75-kW and 3.0-kW groups (both grown at 22.5°C) had respective mean generation times of 13.71 and 13.58 days. The means for the F1 flies from the 2.1-kW group and its control group (both grown at 21.0°C) were respectively 14.61 and 14.64 days. The authors ascribed the 1-day-longer generation time for the latter two groups to the difference in growth temperature, not to the RFEMF exposure.

Brood-size variations were seen among the daily broods from each exposure and control group, so the daily results from each exposure level were pooled, divided by the number of successful cultures, and normalized to the day-1 means for the corresponding control groups. The normalized average F1 brood size on each day of serial mating was presented graphically for each exposure group and its control group. On corresponding mating days, the differences in mean brood size were not significant. Relative infertility was observed between days 5 and 7, but in control as well as RFEMF groups.

The authors noted that since only the heterozygous F1 females would carry an exposed X-chromosome from the male parent, the nonsignificant differences in brood size above may not be an adequate assessment for detrimental effects. However, comparisons of ratios of F1 females to total F1 flies, which should reveal whether RFEMF had damaged the genome of the male parents, yielded no significant differences. In addition, the results for the F2 generations yielded mutations that did not exceed 1%.

Hamnerius et al. (1979) exposed embryos [larvae] of the fruit fly D. *melanogaster* to 2.45-GHz CW RFEMF. The embryos studied were 1-2 hours old and of a sex-linked, genetically unstable stock in which the eye color is light yellow. The mutation sought was somatic, in which a shift in eye pigmentation results in eye sectors with normal red pigmentation clearly visible against the yellow background, a mutation occurring at an early stage of eye development.

For exposure, embryos were immersed in 10 ml of water within a Teflon container, which was placed inside a larger Plexiglas container through which water held at 24.5°C flowed. The larger container was placed at about the start of the far-field region of a horn, at which site the RFEMF level was measured with a power-density meter. SAR was calibrated by measuring the temperature rise in a biological sample due to a 30-second exposure at 900 mW/cm^2 with no water flowing through the larger container; the result was 0.5 W/kg per mW/cm^2. The RFEMF source was a magnetron, so the RFEMF may have been amplitude-modulated at the power frequency for the magnetron.

Embryos were exposed at 100 W/kg (about 200 mW/cm^2) for 6 hours, and control embryos were treated similarly except for exposure. After treatment, the embryos were transferred to vials that contained standard medium and were maintained at 25°C and 75% relative humidity. The survival rate of the flies was determined from the number of male flies that hatched from the embryos, and the percentage of flies having red sectors constituted the mutation frequency. The mean survival rates for exposed and control flies were respectively 83% and 91%, a statistically nonsignificant difference. There were 4 mutations in 7,512 RFEMF-exposed males (0.05%) with a 95% confidence limit [CI] of 0.015-0.136, and 2 mutations in 3,344 control males (0.06%) with a CI of 0.007-0.216, also a nonsignificant difference.

These authors also exposed fly embryos to X-rays as a positive control, and found that 1,000 rad yielded 29 mutations in 1,053 males (2.75%) with a CI of 1.88-4.02. This result led them to conclude that with a sample size of 7,512 males, it would be possible to detect this mutagenic effect at 50 rad (p=0.05).

The authors also noted that exposure to EMS, a chemical mutagen, yielded 444 mutations in 4,859 males (9.14%) at 9.75 mM with a CI of 9.11-10.00.

Possible confounding parameters, such as temperature rise, appear to have been controlled adequately. Also, the significant numbers of mutations observed with the use of positive controls is strong evidence that the mutation-detection methodology was quite satisfactory. Thus, the finding of no RFEMF-induced mutagenic effect in this study is highly credible.

Hamnerius et al. (1985), in an investigation supplemental to Anderstam et al. (1983) and Hamnerius et al. (1979), exposed both S. *typhimurium* bacteria and D. *melanogaster* fruit flies as shown in Table 40 below:

TABLE 40: SALMONELLA & DROSOPHILA EXPOSURE PARAMETERS
[Hamnerius et al. (1985)]

EXPOSURE	SAR (W/kg)	INTENSITY	DUR. (hours)	TEMP. (°C)
		SALMONELLA		
27.12-MHz magnetic	<0.15	21 A/m	6	37.0
27.12-MHz magnetic	22	21 A/m	2.5	25.0
2.45-GHz modulated	130	--	5.7	37.0
3.10-GHz pulsed	90	--	6	37.0
		DROSOPHILA		
27.12-MHz magnetic	<0.05	12 A/m	6	25.0
27.12-MHz magnetic		137 A/m	6	25.0
2.45-GHz modulated		--	6	25.0
3.10-GHz pulsed		--	6	25.0

In the Ames test (Ames et al., 1975, Ames, 1979), chemical mutagens are tested on petri plates with specially constructed mutant strains of S. *typhimurium* selected for sensitivity and specificity to reversion from dependence to independence on histidine. Hamnerius et al. (1985) used an experimentally validated slight modification of this test.

Four strains of Salmonella were grown in nutrient broth, growing cultures were diluted appropriately, and each culture was divided into two parts; one part (10-ml) was exposed as noted above and the other served as an unexposed control. After treatment, 0.1-ml samples were spread on minimal glucose agar plates and incubated at 37°C for 48 hours in darkness before scoring for mutation frequency.

To test for RFEMF-induced cell-division rate or toxicity, the culture samples were diluted by a factor of one million with 0.9% saline, spread on rich agar plates, and incubated at 37°C for 24 hours before scoring for concentrations of viable cells. The results for each strain after each treatment were tabulated as the total number of reversions counted, the mean percentage of mutations relative to controls, the total number of colonies counted, and the mean survival percentage relative to the controls, with their 95% confidence intervals and significance levels.

The data showed that none of the RFEMF exposures significantly increased the numbers of reversions in Salmonella relative to controls; in fact, the number of reversions for one strain following exposure to 2.45 GHz was significantly lower than for controls. Moreover, significant increases in percentage survival were observed for various exposure conditions. Also noticed was a tendency for RFEMF-exposed cells to divide at a higher rate than control cells.

To determine whether the survival-rate increases were thermally induced by the RFEMF, the effect of cultivation temperature on growth and survival was ascertained. Each strain of Salmonella was cultivated at 37.0°C until the culture density was the same as that at the start of RFEMF exposure. Parts of each culture were then transferred to baths at 36.5°C or 37.5 ± 0.1°C and the percentages of survival relative

to those for 37.0°C were measured after growth for at least six generations. The results indicated that the cell concentration during the stationary phase was highest for cells grown at 37.0°C except for one strain, for which 36.5°C yielded the highest growth. Cultures at this stage were no longer in the log phase of growth, but had reached a plateau in number of cells--the stationary phase--which lasted from about 4 hours to at least 7 hours after the temperature shift.

The authors remarked: "The pooled data show that the optimal temperature for growing Salmonella is 37.0°C and that the further increases in cell concentration obtained after electromagnetic field exposure was not due to a change in temperature, since this should have lowered the cell concentration. Consequently, the consistent results obtained after electromagnetic field exposure imply that the increased growth in the exposed cultures is an effect of the electromagnetic fields per se and not secondary to an increase in temperature."

The validity of the point above is unclear because the cultures were supposedly maintained at 37.0 ± 0.3°C during RFEMF exposure. In addition, the authors stated: "However, thermal effects should not be completely ruled out because, according to Blackman et al. (1975), even slight temperature differences (0.2-0.3°C) can affect the growth of Escherichia coli."

For the experiments with Drosophila, a strain was used that is believed to be rendered unstable by inserting a piece of foreign DNA to the right of the white locus. This insertion alters gene expression either spontaneously or to a higher degree by mutagen induction, resulting in eye-pigmentation change in males that are observed by the presence of red sectors in the light-yellow eyes.

Embryos were collected on a yeasted surface of an agar plate. They were washed at age 12 ± 4 hours, and a 0.025-ml batch containing about 4,000 eggs was transferred to 10 ml of water in the exposure container. Samples were then exposed to RFEMF for 6 hours at 25°C with air bubbled through the water to keep the embryos in motion. After exposure, the eggs were transferred to vials that contained standard commeal-yeast-syrup-agar medium, and were kept at 25°C and 75% relative humidity until hatching. Males were screened for the presence of red sectors. Survival was measured by transferring a fixed number of embryos (100 or 200) to vials and counting the number of males hatched in each vial. The results were analyzed for homogeneity of survival and homogeneity of mutation frequencies.

None of the RFEMF exposure conditions altered the mutation frequency or the survival rate of Drosophila embryos significantly. The authors noted that the pooled mutation frequency of the controls was 0.07% (16 among 22,288 males), in good agreement with the expected spontaneous rate. The mutation frequency for methyl methanesulfate (MMS) [the positive-control mutagen used in this study for modified Ames test was 3.5%, a highly significant increase.

Sagripanti et al. (1987) reported the detection of single-strand and double-strand breaks in purified plasmid DNA from exposure to low levels of RFEMF in the range 2.00 to 8.75 GHz, and investigated the role of copper ions in producing such strand breaks. The authors selected E. coli strain HB 101 from which to obtain the samples; that strain contains plasmid pUC8.c2 and was investigated by Edwards et al. (1984, 1985) for microwave absorption. The authors described a sequence of steps to obtain plasmid DNA samples that were purified to ensure the absence of protein contaminants, which was verified spectrophotometrically. Among the steps was incubation of the plasmid DNA with an enzyme to convert it into the linear form related to double-strand breaks. Samples were prepared on agarose gel, subjected to electrophoresis, and stained and photographed for identification.

The samples exposed consisted of 10 μg of plasmid DNA in 28 μl of buffer, with each sample contained within a 1.5-ml micro test tube. The exposures were done by immersing the open end of a coaxial cable as a probe into each sample. The probe itself consisted of the flush open end of the cable, with a solid outer conductor 3.58 mm in diameter and central conductor 1 mm in diameter, both of copper, and with solid dielectric between them. The volume of the probe inserted into the sample was about 2 μl, so it was not considered the dominant heat sink. The attenuation and standing-wave ratio were measured with a dual-directional coupler and a slotted line inserted in the cable between the CW-RFEMF

generator (a Hewlett Packard Model 8616A) and the probe, and the results were used to determine the maximum and minimum SARs. The data indicated that the maximum SAR was about 5 times larger than the minimum SAR, with the true SAR somewhere between the two. The experimental results were referenced to the values of maximum SAR. Other samples were similarly sham-exposed. In addition as controls, samples were sham-exposed with the probe spaced close to but not in contact with each sample.

In the first experiments, samples were sham-exposed or exposed for 20 minutes at 10 W/kg (maximum SAR) to 2.55-GHz RFEMF, a frequency of maximum resonant absorption found in the study by Edwards et al. (1985). The results of 6 experiments showed that the mean number of double-strand breaks in the RFEMF-exposed samples was significantly higher than for the sham-exposed samples. The authors regarded such exposures as nonthermal because of the large surface-to-volume ratio of the samples, thereby ensuring efficient heat dissipation. They also indicated that exposures at levels of about 1000 W/kg were needed to detect any significant temperature rises in the samples.

In other experiments seeking frequency specificity of effect, samples were exposed to 8.75-GHz RFEMF [another frequency previously found by Edwards et al. (1985) to produce maximum resonant absorption by DNA] and to 2.00-GHz, 3.45-GHz, and 7.64-GHz RFEMF, frequencies of minimum absorption found in the same study. The authors remarked that they could not find any variation in double-strand breaks attributable to resonant absorption by the DNA.

For statistical analysis, the data on 12 experiments at the five frequencies above were pooled. The results showed a significantly higher mean percentage of double-strand breaks for the RFEMF-exposed samples than for the corresponding sham-exposed samples. However, the mean percentage of double-strand breaks for the sham-exposed samples was also significantly higher than for the control samples, for which the copper probe was close to the sample but not in contact with it. Moreover, with the probe covered with a thin plastic coating, the difference between sham-exposed and control samples vanished, and no strand breaks were detected in RFEMF-exposed samples.

In still other experiments, samples were incubated in either cupric or cuprous chloride, or in the storage buffer (no copper ions present), and were neither RFEMF-exposed nor sham-exposed. The results mimicked the strand breaking seen with RFEMF exposure but only for incubation in cuprous chloride. On the basis of linear damage increases with RFEMF exposure duration, the authors concluded that the presence of cuprous chloride caused the strand breaking, and that the RFEMF increased the effect.

Perhaps not directly relevant to this study, Gabriel et al. (1987) and Foster et al. (1987) were unable to confirm the findings of resonances by Edwards et al. (1985), primarily citing likely artifacts and lower sensitivity in the RFEMF measurement techniques used by the latter. In any case, if the findings of Sagripanti et al. (1987) regarding the role of cuprous oxide are taken at face value, it is difficult to relate them to potential deleterious effects in humans exposed to RFEMF.

5.3 SUMMARY OF RFEMF AND CANCER IN MICROORGANISMS AND FRUIT FLIES

The findings on mutagenesis and genotoxicity in microorganisms and fruit flies are summarized in Table 41 (A through E).

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Blackman et al. (1976)	The authors investigated whether exposure of cultures of the WWU strain of Escherichia coli (E. coli) to RFEMF would induce mutations therein. This strain was selected because it requires arginine (and other constituents) in the nutrient medium for growth. Cells of that strain that grow in the absence of any of the constituents are regarded as mutants.	Cultures in logarithmic phase were grown on plates that either had media containing arginine or did not. Cells were exposed at 35°C to 1.70-GHz at 88 V/m (2.05 mW/cm^2) or 2.45-GHz RFEMF at 10 or 50 mW/cm^2 (15 or 70 W/kg), mostly for 3.5 hours, to include one or more full cycles of DNA replication.	Growth in the total cell populations in arginine-containing and arginine-lacking media during RFEMF- and sham exposure were expressed as mean numbers of cell doublings relative to the initial populations, and were compared. Compared also were the concentrations of arginine-independent mutants in these populations. The difference in each endpoint between the cultures exposed to the 1.7-GHz RFEMF and sham-exposed cultures was nonsignificant. This was also found for the cultures exposed to 2.45-GHz RFEMF at 10 mW/cm^2 (15 W/kg) for 3.5 hours or at 50 mW/cm^2 (70 W/kg) for 3.2 hours. However, exposure to 2.45-GHz RFEMF at 50 mW/cm^2 (70 W/kg) for 4.0 hours instead of 3.5 hours yielded a significant increase in the cell doublings and a significant decrease in the concentrations of mutant cells.	The authors, citing a previous study (Blackman et al., 1975)], regarded the results with 2.45-GHz at 50 mW/cm^2 (70 W/kg) for 4.0 hours solely as a thermal effect. They concluded that exposures to the RFEMF at either frequency produced no mutagenic activity.
Dutta et al. (1979)	Sought were mutagenic effects of RFEMF in three prokaryotic-cell strains (without true nuclei), TA-1535, TA-100, and TA-98, of Salmonella typhimurium bacteria. Also sought were mutagenic effects in D4 Saccharomyces cerevisiae, a diploid eukaryotic yeast (having true nuclei) of more genome complexity than prokaryotic bacterial cells.	Yeast cells in log phase were exposed for 2 hours at 30°C to 2.45-GHz CW RFEMF (S-band) at 20 mW/cm^2. Other cells were exposed for 2 hours at 29°C to pulsed RFEMF in the range 8.5-9.6 GHz (X-band) at 1000 pps, duty cycle 0.001, with average power densities up to 45 mW/cm^2. Control cultures were similarly sham-exposed. Bacteria cells in log phase were exposed at 37°C to the same S-band levels or at 35°C to the same X-band levels but for 1.5 rather than 2 hours. The SARs for both types of cell were about 40 W/kg.	After exposure, yeast cells were incubated for 72 hours and then scored for the numbers of cells converted to adenine independence and tryptophan independence per 100,000 surviving cells. Similarly, post-exposure bacterial cells were incubated for 48 hours and then scored for the total number of colony-forming units per ml and the number of revertant cells per hundred million survivors. The ratio of each endpoint value after RFEMF exposure to the corresponding value after sham exposure, called the Genetic Activity Index (GAI) was calculated for both, with 1.0 or below signifying no effects. The GAIs for the yeast cells and bacterial cells exposed to the S-band RFEMF showed little genetic changes relative to controls. The yeast-cell GAIs, tabulated at 9.0-GHz for adenine and tryptophan versus power density, were all well below 2.0 except adenine for 45 mW/cm^2 at 9.4 GHz (GAI=2.17). The GAIs for the respective endpoints of each of the three bacterial strains were also well below 2.0.	The authors noted that GAI values that do not exceed 1.0 signify no RFEMF-induced mutagenesis, values between 1.0 and 2.0 are within the normal fluctuation range, those between 2.0 and 3.0 are "suspect", and those exceeding 3.0 definitely indicate mutagenesis. On this basis, they concluded that exposures of the yeast or bacteria to the RFEMF did not reliably produce genetic changes, noting that all of the GAIs were below 3.0. However, they did not present any statistical treatment, or clarify why large variations were seen in the results for the various sham-exposed samples. Also not clear is the biological significance, if any, of the number of GAIs that were well below 1.0. Thus, although the findings of this study were negative, non-RFEMF factors may have been present in the experiments.

TABLE 41A: MUTAGENESIS AND GENOTOXICITY IN MICROORGANISMS AND FRUIT FLIES

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Dardalhon et al. (1979)	The authors sought possible mutagenetic effects of RFEMF and elevated temperature on the yeast Saccharomyces *cerevisiae*. A diploid strain of S. *cerevisiae* having two alleles distinguishable in color was used to study cell survival and induction of mitotic recombination, or "petite" cytoplasmic mutations. Testing for zygote formation was done using two haploid strains, both genetically deficient in synthesizing certain amino acids, but opposite mating type with complementary new-growth characteristics.	Cells at 20°C grown on agar in open petri dishes were exposed from above for 180 or 330 minutes to 70.5-GHz or 73-GHz CW RFEMF [in the millimeter-wave region] or were sham-exposed as controls. The power densities ranged up to 60 mW/cm^2 at sites 2 mm or 10 mm from the face of a horn [in the near field]. Exposure to 60 mW/cm^2 at baseline 20°C was shown to increase sample temperature by no more than 2-3°C. For comparison, other dishes were held at 30°C, 37°C, 42°C, 47°C, and 52°C for 330 minutes.	The ratios of the survival data of diploid-strain cells exposed to 70.5-GHz or 73-GHz RFEMF at 15 or 60 mW/cm^2 at the 10-mm site for up to 180 minutes to the corresponding data for the controls versus treatment durations changed little from unity with time. [Data for the 2-mm site were not given.] With the color-change technique, the percentages of altered colonies were found to be "practically nil" after exposures under the four conditions above, indicating that the RFEMF had no effect on nuclear DNA. The results for both exposure sites were also negative for induction of cytoplasmic "petite" mutations in the two haploid strains, indicating that the RFEMF had no adverse effects on mitochondrial DNA. Conventional heating at up to 47°C also had no effect on relative survival ratios, percentages of altered colonies, or "petite" mutations, but decreases in survival percentages and increases in percentages of altered cell colonies and "petite" mutations were observed at 52°C.	The finding that under the stated conditions, exposure to millimeter waves does not induce lesions or genetic effects in cellular DNA of S. *cerevisiae*, in contrast with intense conventional heating, is scientifically credible. However, the authors did not provide any statistical treatment of the results, thereby diminishing confidence in their findings. Based on their results, the authors remarked that the reported findings of Webb and Dodds (1968) on the inhibition of bacterial growth by exposure to 136-GHz RFEMF cannot be explained by direct [nonthermal] action of RFEMF on cellular DNA, a comment supported by independent findings of other investigators.
Dardalhon et al. (1981)	Sought were possible effects of 9.4-GHz and 17-GHz RFEMF on various ultraviolet-sensitive strains of procaryote E. coli and of eucaryote S. *cerevisiae*. Also investigated further were possible effects in the range 70-75 GHz.	Most exposures were for 30 minutes. The sites for 9.4-GHz exposures were 5 mm and 3 cm from a horn. Normalized SARs at 5 mm were 0.38 W/kg per mW/cm^2. The same sites were used for 17 GHz, but from an open waveguide. The SARs at 5 mm were 0.56 W/kg per mW/cm^2. (Those at 3 cm were not given.) For 70-75 GHz, the 2-mm site in the previous study was used, with SARs of 0.15 W/kg per mW/cm^2. As positive controls, cell samples were exposed to 254-nm ultraviolet radiation.	Exposure of E. coli, including several strains deficient in DNA repair, one wild-type DNA-repair-proficient strain, and a mutant strain requiring tryptophan for growth, to the different RFEMF frequencies yielded non-significant differences in survival rates relative to sham-exposed cells or in the ratios of mutations of tryptophan-dependent cells to tryptophan independence. Several haploid and diploid strains of S. *cerevisiae* were studied, each for specific endpoints, including survival rates, effects on DNA repair, genetic alterations, and sporulation effects. The survival rates for cells exposed to RFEMF also did not differ significantly from those for sham-exposed cells. The percentages of cytoplasmic petite mutations in the haploid strain after RFEMF exposure did not differ significantly from the percentage of spontaneous mutations. Sporulation efficiencies, assessed by exposing cells of one diploid strain for 48 hours during sporulation or another strain for 72 hours during meiosis, did not differ significantly from those for sham exposure.	As for the previous study, the lack of statistical treatment of the results diminishes confidence in the findings.

TABLE 41B: MUTAGENESIS AND GENOTOXICITY IN MICROORGANISMS AND FRUIT FLIES (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Anderstam et al. (1983)	These authors investigated whether RFEMF is mutagenic for E. coli and S. *typhimurium* (using a total of 11 strains), but at 27.12 MHz, 2.45-GHz, and 3.1 GHz.	Cells in 10-ml volumes were exposed between parallel plates to a 27.12-MHz electric field at 72 V/m (SARs 3 W/kg), or a 27.12-MHz magnetic field in a Helmholtz coil at 20 A/m (SARs about 20 W/kg). Other cells were exposed to far-field, 2.45-GHz RFEMF amplitude-modulated at 100 Hz or pulsed 3.07-GHz RFEMF, both at about 0.5 W/kg per mW/cm^2. Exposure durations were in the range 1-7 hours.	The tabulated results of the various RFEMF treatments of each strain were mixed, e.g., some strains given some RFEMF treatments exhibited higher growth and others lower growth than their respective controls. Many of these changes were nonsignificant, but the overall trend was toward RFEMF-induced increase in growth. The authors could not ascribe the growth changes solely to temperature increases, stating that: "...changes of temperature in unexposed samples by 0.5°C certainly provoked changes of the growth rate, but these changes were smaller than observed after microwave exposure." However, another finding was that the differences in mutant counts (both increases and decreases) induced by RFEMF were mostly nonsignificant.	The authors tabulated considerable quantities of data and discussed their statistical treatments and interpretations thereof, based on a variety of considerations difficult to assess, mostly because the results were mixed, as noted previously. One overall conclusion of theirs was that risk of cancer and of heritable damage from occupational exposure to RFEMF in Sweden is less than one case per year. However, not clear is whether they had used UV or any other known mutagenic agent alone as a positive control, to assess the adequacy of their methodology, and specifically whether significant non-RFEMF factors were present.
Pay et al. (1972)	Sought were mutagenic effects in Drosophila *melanogaster* flies from exposing males to 2.45-GHz RFEMF, mating them with unexposed female flies, and examining the first and second generations of flies for mutations.Pay et al. (1972)	After eclosion, 3 groups of five Oregon-R, wild-type male flies, each group within an acrylic capsule, were exposed to 2.45-GHz RFEMF in the near field of a horn at forward powers of 2.1, 2.75, or 3.0 kW for 45 minutes. Control groups were sham-exposed. The males of each group were then mated daily for 15 days with two new virgin Muller-5 females to yield first-generation (F1) flies. The adult F1 broods were counted and sexed each day, and 45 male-female pairs from exposed males and 15 male-female pairs from sham-exposed males therefrom were mated to yield second-generation (F2) flies.	Graphs of mean F1 brood sizes versus successive days of serial mating indicated no significant differences for each mating day between the groups RFEMF-exposed at each level and their sham-exposed groups. Comparisons of ratios of F1 females to total F1 flies, which should reveal whether the RFEMF had damaged the genome of the male parents, also yielded no significant differences. On day 17, F2 groups were examined for wild-type males. Their presence would rule out the possibility of sex-linked lethal recessive mutagenesis on the X-chromosome of the parent males exposed to the RFEMF, and such a recessive mutation would cause lethality in F2 hemizygous wild-type males (having only one of a pair of genes for a specific trait). The F2 generations yielded mutation numbers that did not exceed 1%, the stated level of detection, and no lethal mutations were observed.	Although their results were negative, the authors remarked that they could not rule out with certainty the possibility of a short-lived RFEMF-sensitive stage during early spermatogenesis, based on the sensitivity of sperm to X-rays during pre-meiotic and early meiotic stages. However, there is no experimental evidence of direct (not heat-related) effects of RFEMF on sperm.

TABLE 41C: MUTAGENESIS AND GENOTOXICITY IN MICROORGANISMS AND FRUIT FLIES (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Hamnerius et al. (1979)	Studied were effects of 2.45-GHz CW RFEMF in Drosophila *melanogaster* (fruit-fly) mbryos from a genetically unstable, sex-linked stock having a light-yellow (zeste) eye color. The mutation sought was somatic, in which a shift in eye pigmentation results in eye sectors with normal red pigmentation clearly visible against the yellow background.	Embryos 1-2 hours old, immersed in 10 ml of water within a container placed inside a larger container through which flowed water at 24.5°C, were exposed to the RFEMF at 100 W/kg (about 200 mW/cm^2) for 6 hours. Control embryos were similarly sham-exposed. As positive controls, embryos were also exposed to X-rays at 1,000 rad and to the chemical mutagen EMS at 9.75 mM.	The mean survival rates for exposed and control flies, determined from the number of male flies that hatched, were respectively 83% and 91%, a nonsignificant difference. There were 4 mutations (flies having red sectors) in 7,512 RFEMF-exposed males (0.05%) and 2 mutations in 3,344 control males (0.06%), also a nonsignificant difference. The X-ray exposures yielded 29 mutations in 1,053 males (2.75%), a result that led the authors to conclude that, with the 7,512 male sample size, it would be possible to detect this mutagenic effect at 50 rad (p=0.05). The results with EMS were 444 mutations in 4859 males (9.14%).	The RFEMF source was a magnetron, so the microwaves may have been amplitude-modulated at the frequency of the magnetron's power supply. Possible confounding factor, appear to have been controlled adequately. Also, the significant numbers of mutations observed in the positive controls is strong evidence that the mutation-detection methodology was quite satisfactory. Thus, the finding of no RFEMF-induced mutagenic effect in this study is highly credible.
Hamnerius et al. (1985) [Supplemental investigation to Anderstam et al. (1983) & Hamnerius et al. (1979)]	The authors further sought possible mutagenic effects on Salmonella bacteria and Drosophila fruit flies from exposure to 27.12-MHz magnetic fields, 2.45-GHz RFEMF, and 3.10-GHz pulsed RFEMF (but not to 27.12-MHz electric fields) at levels and durations comparable to those used in their previous studies.	Four specific strains of Salmonella sensitive to reversion from histidine dependence to histidine independence were studied. After several dilution and incubation steps, samples of each culture were exposed to RFEMF and others were sham-exposed. Studied were 12-hour-old Drosophila embryos of a strain rendered unstable by inserting a piece of foreign DNA such that exposure to a mutagen increases the number of male flies that have red sectors in yellow eyes. Embryo samples were exposed to RFEMF for 6 hours at 25°C.	The Salmonella data showed that none of the RFEMF exposures significantly increased the numbers of reversions relative to controls; in fact, the number of reversions for one strain following exposure to 2.45 GHz was significantly lower than for controls. Moreover, significant increases in percentage survival were observed for various exposure conditions. Also noticed was a tendency for RFEMF-exposed cells to divide at a higher rate than control cells. Other experiments led the authors to suggest that the observed increases in percentage survival were a direct effect of the RFEMF rather than thermally induced. The Drosophila data indicated that none of the RFEMF exposure conditions significantly altered the mutation frequency or the survival rate. The authors remarked that the pooled mutation frequency of the controls was 0.07% (16 among 22,288 males), in good agreement with the expected spontaneous rate. By contrast, the mutation frequency for methyl methanesulfate (MMS), the positive-control mutagen used in this study, was 3.5%, a highly significant increase.	The Salmonella data showed that none of the RFEMF exposures significantly increased the numbers of reversions relative to controls; in fact, the number of reversions for one strain following exposure to 2.45 GHz was significantly lower than for controls. Moreover, significant increases in percentage survival were observed for various exposure conditions. Also noticed was a tendency for RFEMF-exposed cells to divide at a higher rate than control cells. Other experiments led the authors to suggest that the observed increases in percentage survival were a direct effect of the RFEMF rather than thermally induced. The Drosophila data indicated that none of the RFEMF exposure conditions significantly altered the mutation frequency or the survival rate. The authors remarked that the pooled mutation frequency of the controls was 0.07% (16 among 22,288 males), in good agreement with the expected spontaneous rate. By contrast, the mutation frequency for methyl methanesulfate (MMS), the positive-control mutagen used in this study, was 3.5%, a highly significant increase.

TABLE 41D: MUTAGENESIS AND GENOTOXICITY IN MICROORGANISMS AND FRUIT FLIES (CONTINUED)

Authors	Effects Sought or Examined	Presumed or Actual Exposure & Treatment	Effects Reported	Notes & Comments
Sagripanti et al. (1987)	The authors used agarose gel electrophoresis to detect single-strand and double-strand breaks in purified plasmid DNA (derived from E. coli) from exposure to low levels of RFEMF in the range 2.00 to 8.75 GHz, and investigated the role of copper ions in producing such strand breaks.	Samples of plasmid DNA in buffer, within a 1.5-ml micro test tube, were exposed to the RFEMF by immersing an open-ended copper coaxial probe into the samples, with the other end of the cable coupled to an RFEMF generator. A directional coupler and slotted line between the probe and generator were used for measuring attenuation and standing-wave ratio. Measurements yielded a maximum SAR about 5 times larger than the minimum SAR, with the true SAR somewhere between them, so the results were referenced to the maximum SAR.	Smples exposed for 20 minutes at 10 W/kg (maximum SAR) to 2.55-GHz RFEMF, a frequency of maximum resonant absorption found in the study by Edwards et al. (1985) yielded mean numbers of double-strand breaks significantly higher than for the sham-exposed samples. The authors regarded such exposures as nonthermal because of the large surface-to-volume ratio of samples, thus ensuring efficient heat dissipation. They also indicated that exposures at levels of about 1000 W/kg were needed to detect any significant temperature rises in the samples. Samples were exposed to 8.75-GHz RFEMF [another frequency previously found by Edwards et al. (1985) to produce maximum resonant absorption by DNA] and to 2.00-GHz, 3.45-GHz, and 7.64-GHz RFEMF, frequencies of minimum absorption found in the same study. The pooled data from 12 experiments at these frequencies showed a significantly higher mean percentage of double-strand breaks for the RFEMF-exposed than the sham-exposed samples. Comparisons of samples incubated in cupric chloride, cuprous chloride, or the storage buffer (with no copper ions present), but neither RFEMF- nor sham-exposed yielded results that mimicked the strand breaking seen with RFEMF exposure but only for incubation in cuprous chloride. Based on linear increases of damage with exposure duration, the authors concluded that the presence of cuprous chloride caused the strand breaking, and that the RFEMF increased the effect.	The authors also reported that the mean double-strand breaks for the sham-exposed samples were also significantly higher than for control samples for which the copper probe was close to the samples but not in contact with them. Moreover, with the probe covered with a thin plastic coating, the difference between the sham-exposed and control samples vanished, and no strand breaks were detected in RFEMF-exposed samples. Perhaps not directly relevant to this study, both Gabriel et al. (1987) and Foster et al. (1987) were unable to confirm the findings of resonances by Edwards et al. (1985), primarily citing likely artifacts and lower sensitivity in RFEMF measurement techniques used by the latter. In any case, if the findings of Sagripanti et al. (1987) on the role of cuprous oxide are taken at face value, it is difficult to relate them to potential deleterious effects in humans exposed to RFEMF.

TABLE 41E: MUTAGENESIS AND GENOTOXICITY IN MICROORGANISMS AND FRUIT FLIES (CONCLUDED)

6 UNRESOLVED ISSUES

The potential carcinogenic effects of RFEMF have been assessed from peer-reviewed studies published in the scientific literature. The preponderance of scientifically credible evidence indicates that there is no association between exposure to RFEMF and the incidence of promotion of cancer, even at levels that produce temperature elevations in mammals that exceed their metabolic regulatory abilities. Nevertheless, there are several basic uncertainties, summarized below, regarding biological effects of RFEMF in general.

(1) Many of the epidemiologic studies on possible bioeffects of RFEMF were extensive and well done, but contained defects or uncertainties in varying degrees, such as imprecise assignment of individuals to exposure and control groups; difficulties in obtaining accurate medical records, death certificates, or responses to health questionnaires for individuals included in both the exposure and control groups; and most important, the large uncertainties about the frequencies, levels, and exposure durations for those selected for inclusion in exposure groups and the amount of exposure received by those selected for inclusion in control groups.

(2) Applying results on laboratory animals to humans, though essential, is an expedient that contains fundamental problems and uncertainties due to the basic differences between humans and other species. Investigations with nonhuman primates may narrow some of the interspecies gaps considerably, but at costs that are often prohibitive. Thus, major reductions in such uncertainties seem unlikely in the near future.

(3) The results of many investigations indicate the existence of threshold RFEMF levels for various bioeffects, thus providing confidence that exposure to levels appreciably below the thresholds are most unlikely to be deleterious. However, most experimental data that indicate the existence of thresholds were obtained by use of single or repetitive exposures of relatively short durations. Although it is difficult to conceive of mechanisms by which RFEMF exposures are cumulative at well below threshold values over a long time, very few investigations have been done that involve essentially continuous exposure of animals to low-level RFEMF (below threshold levels or those that can cause significant heating) during most of their lifetimes. The high costs of such chronic studies and the low probability that any positive effects will be found are major reasons why such studies are not given high priority by funding agencies.

(4) Regarding the basic mechanisms of interaction between RFEMF and various biological entities, many important investigations have been done that involved exposure of cells and subcellular structures and constituents in vitro to relatively low levels of RFEMF. Although some effects on such entities have been reported and were characterized as nonthermal, such findings have not been confirmed in independent studies. Nevertheless, even if such findings are accepted at face value, the gap between such positive effects and possibly hazardous effects on intact humans or animals from exposure to such RFEMF levels is enormous. Factors such as large body masses, penetration depth and internal field distributions, and changes in body orientation during exposures to RFEMF in vivo can vastly moderate such interactions or remove them entirely. Moreover, the life processes per se are extremely complex. For these reasons, this gap is not likely to be reduced to any great extent.

7 MISCONCEPTIONS

Popular media often do not distinguish between RFEMF (nonionizing radiation) and ionizing radiation, so concern is frequently raised in the general public, with no scientific basis, that RFEMF can give rise to hazardous effects known to be caused by ionizing radiation. In essence, any quantum of ionizing radiation absorbed by a molecule yields up enough energy to expel an electron from the molecule (ionize it), leaving it positively charged and thus strongly enhancing the interactions of the molecule with its neighbors. Such interactions can alter the functions of biological molecules fundamentally and irreversibly in living organisms.

By contrast, the energy in a quantum of RFEMF is so much smaller than in a quantum of ionizing radiation that the primary effect of RFEMF quanta is to agitate molecules rather than ionize them. The absorption of RFEMF quanta at high rates (in large numbers per unit time) is necessary to produce physiologically significant heat. Moreover, such RFEMF molecular agitation begins to diminish immediately on cessation of exposure.

It is also necessary to distinguish between an effect and a hazard. For example, a person's metabolism can be increased harmlessly by mild exercise. Analogously, an effect produced at RFEMF intensities that yield heat that can be easily accommodated within the thermoregulatory capabilities of an individual may not necessarily be deleterious. Also, any effects produced thereby are generally reversible. However, the thermoregulatory capabilities of any given species may be exceeded at high RFEMF intensities, so compensation for such effects may be inadequate. Thus, exposure at such intensities can cause thermal distress or even irreversible thermal damage.

It is not scientifically possible to guarantee that exposure to RFEMF (or any other agent) that does not cause deleterious effects for relatively short exposures at low levels will result in the appearance of deleterious effects many years in the future. Primarily because of funding limitations, relatively few studies have been done in which laboratory animals were exposed to RFEMF during virtually their entire lifetimes. However, as noted previously, the experimental data from a number of experimental studies indicate the existence of threshold levels for various RFEMF bioeffects, so chronic exposures to levels well below such thresholds (and particularly below current RFEMF-exposure guidelines) are most unlikely to affect human health.

8 OVERALL CONCLUSION

Based on the foregoing analyses of the epidemiologic/occupational studies, the in vivo and in vitro studies with non-human mammals, and the mutagenetic experiments with microorganisms and fruit flies, there is no scientifically valid basis for the existence of a linkage between cancer incidence or promotion and RFEMF exposure.

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